THE
AMERICAN
SURGEON

Vol. 23, No. 7 July, 1957

A PHOTOGRAPHIC STUDY OF THE SUPERIOR AND INFERIOR LARYNGEAL NERVES AND THE SUPERIOR AND INFERIOR THYROID ARTERIES

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A detailed knowledge of the surgical anatomy of the superior and inferior laryngeal nerves and their corresponding thyroid arteries is essential for surgeons who undertake surgical procedures upon the thyroid gland. In order to have a more thorough understanding of these structures, we undertook to dissect, study, and photograph the thyroid compartment of the anterior neck of 50 recently embalmed adult bodies. The photograph of each dissection was made through the use of a portrait attachment on an Exacta camera equipped with kodachrome film. The superior and inferior laryngeal nerves in each dissection were painted yellow with a rubber-base paint. The corresponding arteries were painted red with the same type paint. The technic of preparing freshly dissected specimens for photography was suggested by Doctor W. Henry Hollinshead.[4] The work was done in the pathology laboratory of Presbyterial Hospital, Denver, through the courtesy of Doctor Alexis Lubchenco.

The study is somewhat defective since descriptive terms used to indicate minor variations were frequently confusing. Because of this only the dissection records in which the relationships were unquestionably clear, are included in the statistical tabulations.

The inferior laryngeal nerve is regarded in most anatomic descriptions as a single trunk nerve which passes upward along the tracheoesophageal groove, and enters the larynx at the region of the cricothyroid membrane. In January of 1954 Rustad[9] described the inferior laryngeal nerve as a divided nerve which often enters the larynx in many diverse and unpredictable branching patterns. Forty-three per cent of the specimens in this series had nerves with from 2 to 6 extra-

Presented during the Tucson Assembly of The Southwestern Surgical Congress, April 16–18 1956, Tucson, Arizona.
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laryngeal branches. At first our interest was largely centered about the relationship of the inferior laryngeal nerve to the inferior thyroid artery. Because of Rustad's work, a more detailed study of each inferior laryngeal nerve was made during the dissection of our last 25 cadavers. In this group (table 1) the inferior laryngeal nerve was found to be a single trunk nerve in 42 per cent and a divided nerve in 58 per cent of the 50 dissections. Seventeen had 2 branches, 7 had 3 branches, 4 had 4 branches, none were found with 5 branches but 1 was found with 6 branches. Figure 1 demonstrates a specimen in which 3 extra-laryngeal branches were found, and figure 2 shows a specimen with 4 extra-laryngeal branches. It is apparent that a significant number of the patients encountered at surgery can be expected to have more than one inferior laryngeal nerve trunk.

INFERIOR NERVE BRANCHES			
	DISSECTIONS	SINGLE TRUNK	2-6 BRANCHES
LEFT	25	10	15
RIGHT	25	11	14
TOTAL	50	21	29
%	100	42	58

TABLE 1

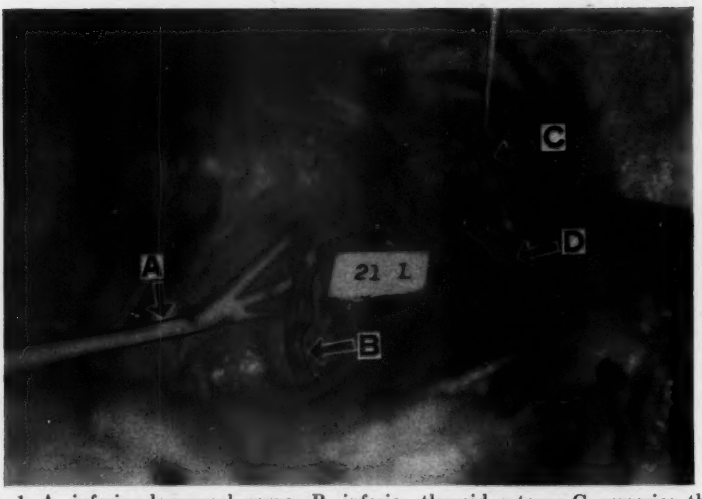

FIG. 1. A, inferior laryngeal nerve; B, inferior thyroid artery; C, superior thyroid artery; D, superior laryngeal nerve.

Berlin and Leahey,[1] Fowler and Hanson,[3] and later Nordlund[8] all called attention to relationships between the inferior thyroid artery and the inferior laryngeal nerve. The work of these men indicates that there is no relationship between the artery and the nerve which may be constantly depended upon in the performance of thyroid surgery.

In our study the relationship of the inferior laryngeal nerve to the inferior thyroid artery (table 2) was clearly recorded in 48 cadavers giving a total of 96 dissections. In 50 per cent of these dissections the nerve, either as a single trunk or a divided structure, was medial to all parts of the inferior thyroid artery, and in 10 per cent it was lateral. In 40 per cent, however, a mixed relationship oc-

FIG. 2. A, inferior thymoid artery; B, 4 extra laryngeal branches inferior laryngeal nerve; C, tracheal branch.

NERVE TO ARTERY POSITION				
	DISSECTIONS	MEDIAL	LATERAL	MIXED
LEFT	48	32	1	15
RIGHT	48	16	9	23
TOTAL	96	48	10	38
%	100	50	10	40

TABLE 2

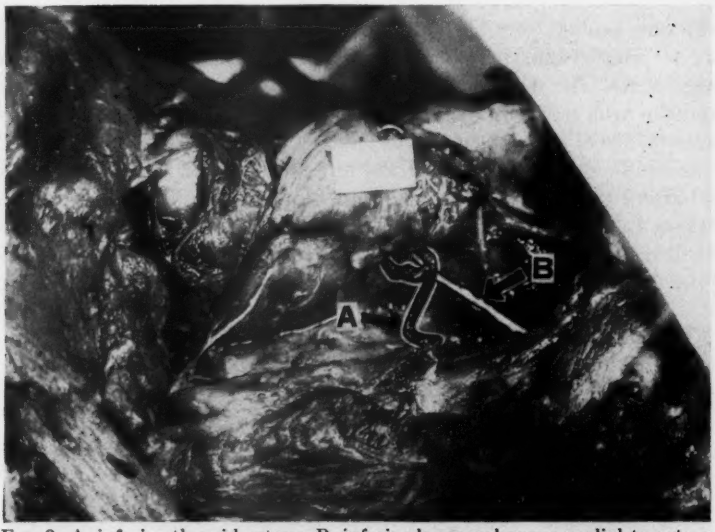

FIG. 3. A, inferior thyroid artery; B, inferior laryngeal nerve medial to artery

FIG. 4. A, inferior thyroid artery; B, inferior laryngeal nerve lateral to inferior thyroid artery.

curred in which one or more branches of the nerve was lateral or medial to one or more branches of the artery, being at the same time in the opposite relationship to other branches of the artery. Figure 3 illustrates a medially placed nerve, and figure 4 is an example of the one lateral nerve out of 48 dissections on the left. Figure 5 demonstrates the mixed nerve-artery relationship.

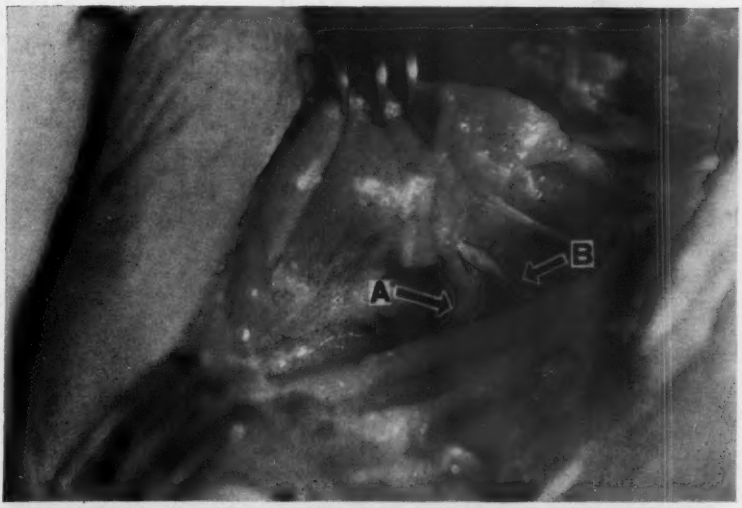

FIG. 5. A, inferior thyroid artery; B, inferior laryngeal nerve a mixed relationship

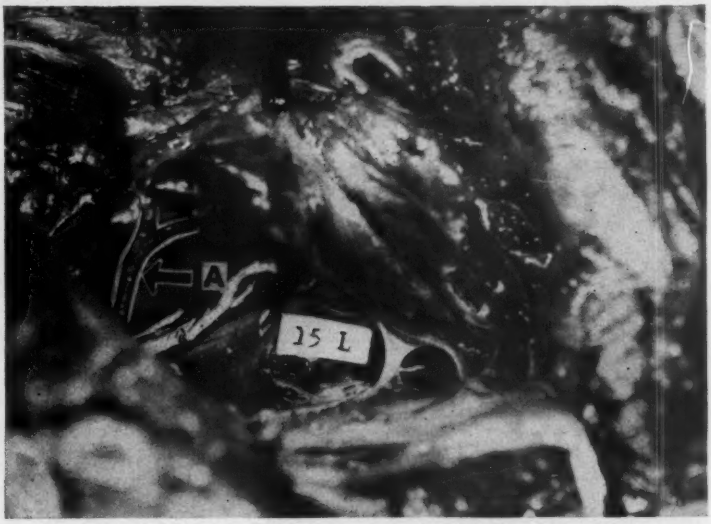

FIG. 6. A, inferior thyroid artery proceeding directly to lower poles

The feasibility of ligating the inferior thyroid artery in selected cases at a point lateral to its dangerous relationship to the inferior laryngeal nerve seemed a matter which would bear anatomic investigation. The extra capsular ligation of the inferior thyroid artery is not a new idea, having been described by de Quervain[2] in 1916.

The artery usually arises as a branch of the thyro-cervical trunk of the sub-

clavian artery. It normally passes upward in the neck posterior to the carotid sheath. At the level of the thyroid gland the artery turns medialward, leaves its position behind the carotid sheath and forms an ascending and descending loop close to the posterolateral border of the gland near the junction of its middle

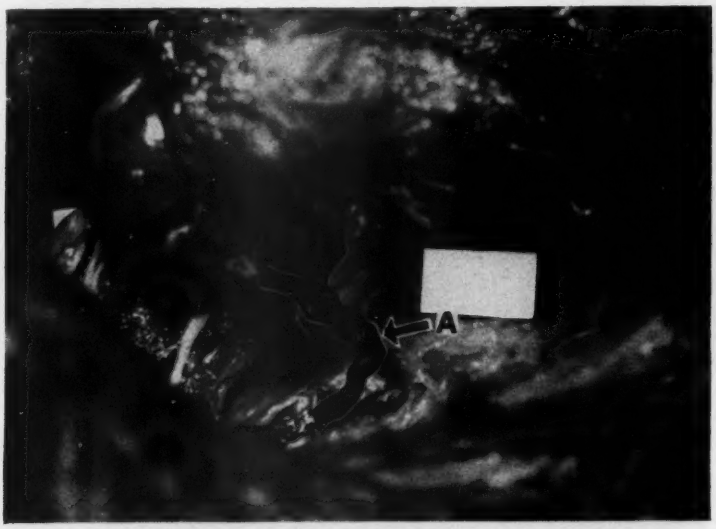

FIG. 7. A, inferior thyroid artery shows loop at middle of gland

FIG. 8. A, inferior thyroid artery showing high loop opposite the superior pole

and lower thirds. It is the presence of this loop of the inferior thyroid artery and the variations in the height to which it ascends which seems to create some difficulty for surgeons who search for the inferior thyroid artery with the idea of ligating it in selected cases.

In our dissections we found that the artery sometimes emerges near the lower pole of the thyroid proceeding directly to the gland in a more or less straight course (fig. 6). Sometimes we found it to emerge at about the middle of the gland looping downward toward the lower pole (fig. 7). Frequently we found it to emerge near the upper pole creating a high loop before descending toward the lower pole (fig. 8). We divided these locations into low, middle, and high and found (table 3) that of 78 adequately recorded specimens 27 per cent of the arteries were in the low group, 47 per cent were in the middle group, and 25 per cent in the high group. In an additional specimen the inferior thyroid artery was absent. In this instance the superior thyroid artery was larger than normal.

Negus[7] started the controversy about the function of the superior laryngeal nerve in 1929, by indicating that although the intrinsic muscles of the larynx are largely supplied by the inferior laryngeal nerve, the circo-thyroid muscle is

INFERIOR ARTERY POSITION					
	DISSECTIONS	LOW	MIDDLE	HIGH	ABSENT
LEFT	40	9	18	12	1
RIGHT	38	12	19	7	0
TOTAL	78	21	37	19	1
%	100	**27**	**47**	**25**	1

TABLE 3

SUPERIOR LARYNGEAL NERVE				
	DISSECTIONS	IN DANGER	NO DANGER	QUESTIONABLE
LEFT	37	25	7	5
RIGHT	32	22	7	3
TOTAL	69	47	14	8
%	100	**68**	**20**	**12**

TABLE 4

innervated by the external division of the superior laryngeal nerve and the interarytenoid muscle receives some fibers from the internal division of the superior laryngeal nerve. Lemere[6] in 1932 was convinced that the internal division of the superior laryngeal nerve is entirely sensory. In 1948 King and Gregg[5] provided

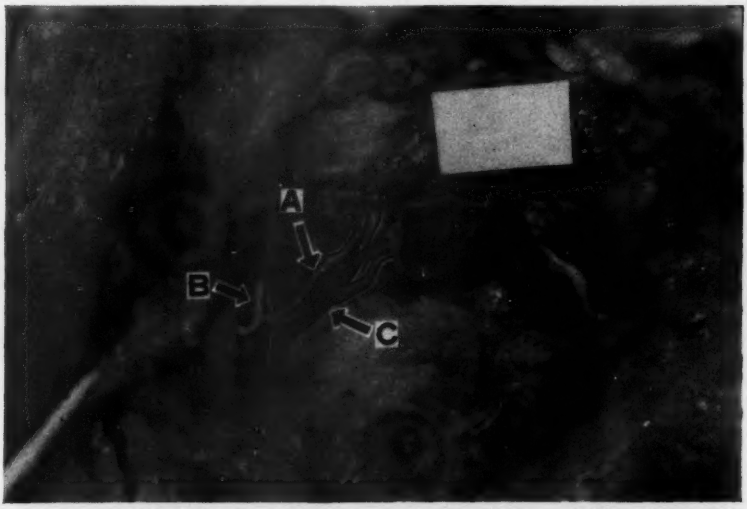

FIG. 9. A, external division superior laryngeal nerve in close relationship to superior thyroid artery; B, internal division of superior laryngeal nerve; C, superior thyroid artery.

FIG. 10. A, superior thyroid artery; B, external division of superior laryngeal nerve showing an out-of-danger relationship with the superior thyroid artery.

support for Lemere's thesis by demonstrating that faradic stimulation of the internal division of the superior laryngeal nerve of the dog uniformly caused no contraction of the interarytenoid muscle and that similar stimulation of the external division of the superior laryngeal nerve caused contraction of the cricothyroid muscle alone. In 1930 Nordlund[3] noted that it was easy to injure the superior laryngeal nerve during the ligation of the superior thyroid artery and believed that postoperative voice changes such as weakness, roughness, easy fatiguability, and decreased range might well occur because of cricothyroid paralysis. In our study the position of the superior laryngeal nerve was recorded in 69 dissections (table 4). In all of these the internal division was observed to be several centimeters above the superior pole of the thyroid gland or above the point of usual superior thyroid artery ligation. The external division, however, was often closely associated with the superior thyroid artery. We considered it to be in danger, so far as the ligation of the superior thyroid artery was concerned, in 68 per cent of these specimens and out of danger in 20 per cent. The external division was not positively identified by histologic section in 8 specimens or 12 per cent of this group. (Figure 9) illustrates an in-danger relationship, and figure 10 shows a nerve which is apt to be out of danger during ligation of the superior thyroid artery.

No particular attempt was made in this study to search for all parathyroid glands, but it was found that one parathyroid was regularly located close to one of the terminal branches of the inferior thyroid artery (fig. 11) behind the thyroid gland but in the areolar tissue of the false capsule about midway between the tip of the lower pole and the mid point of the gland. Our best method of locating it was to follow the branches of the inferior thyroid artery. We were unable to

FIG. 11. A, one branch inferior thyroid artery showing close relationship to parathyroid; B, parathyroid gland; C, inferior laryngeal nerve.

positively identify a parathyroid in 14 dissections. There is no doubt that more diligent search in each instance would have revealed its presence. It was our opinion, however, that a cursory search for parathyroid tissue at the time of thyroid surgery will be rewarding in most instances if the branches of the inferior artery are identified and followed to the gland.

SUMMARY

A dissection of the thyroid region of the neck was carried out in 50 cadavers. The superior and inferior thyroid arteries and the superior and inferior laryngeal nerves were isolated and painted red and yellow respectively. The specimens were then photographed. An accurate recording of the anatomic relationship of these structures was made in the majority of specimens. The results of these findings have been presented.

CONCLUSIONS

The external division of the superior laryngeal nerve is often in danger during the ligation of the superior thyroid artery because of its frequently close relationship to the artery at the point of usual ligation.

There is no constant relationship between the inferior thyroid artery and the inferior laryngeal nerve. In many specimens a mixed relationship exists between branches of the nerve and the artery.

Ligation of the inferior thyroid artery proximal to its branching requires the understanding that the inferior thyroid artery more often emerges from behind the carotid sheath at a middle or high position rather than at a position directly opposite the lower pole.

The inferior laryngeal nerve often branches extra-laryngeally.

At least one parathyroid gland can be located readily by following the branches of the inferior thyroid artery.

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TISSUE NECROSIS ASSOCIATED WITH INTRAVENOUS NOR-EPINEPHRINE ADMINISTRATION*, †

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Even though well known for its beneficial results in shock-like states, epinephrine has been surpassed by its isomer l-norepinephrine as a pressor agent. Probably no more powerful vasoconstrictor has been identified than l-norepinephrine. Nor-epinephrine, a catechol amine, exerts its effect by producing generalized arterial, capillary and venous constriction, thereby resulting in a marked increase in systemic blood pressure. Because a decreased renal plasma flow occurs in the presence of little or no change in glomerular filtration rate, the filtration fraction rises with a resultant pattern similar to that noted in certain types of essential hypertension.

Where pronounced hypotension, oliguria, increased cardiac work and tachycardia become sustained, the prognosis for life becomes extremely poor unless these undesirable features can be overcome. Nor-epinephrine has proved its capability of reversing many of the pathologic features attendant to peripheral vascular collapse. Despite its lifesaving features, ulceration of the skin and deep subcutaneous structures as a result of the drug's marked vasoconstrictive properties has occurred in our experience.[5] Because of this and the reports of others,[2, 6, 7, 8, 9, 11] we believe further investigation of this problem is warranted.

While reports vary as to the occurrence of local necrosis following subcutaneous and intravenous administration of norepinephrine, our series of cases would seem to leave little doubt as to its destructive ability. In order to demonstrate these features of the drug, the following representative clinical cases are reported in some detail. To further clarify the nature of action of l-norepinephrine, the clinical aspects are further substantiated by animal experimental investigation.

CASE REPORTS

Case 1. Mr. L. M., a 40 year old white man, known chronic alcoholic, was admitted to the hospital in semicoma in June of 1953. The clinical picture was not clear; however, he was believed to be manifesting symptoms following the sudden withdrawal of alcohol. Conservative treatment was instituted; this included the administration of intravenous nor-epinephrine solution 4mg./1000cc into the left antecubital vein because of persistent hypotension.

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† A portion of the experimental section was presented at the annual meeting of the Seattle Surgical Society, January 29, 1954.

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FIG. 1. Diffuse pallor in ante-cubital fossa of patient number 1. Photograph was taken over 5 hours after infiltration of nor-epinephrine solution.

An estimated 100 cc. of this solution infiltrated into the surrounding tissues. Marked blanching was noted immediately and this ischemic response persisted. A photograph was taken 5 hours after the infiltration of nor-epinephrine solution, and the steadfast ischemia is demonstrated in figure 1. The patient did not respond to therapy and died 12 hours after admission.

Case 2. Mrs. L. W. a 27 year old unmarried Para II, Gravida III, white woman, was first seen in our hospital on the date of admittance Jan. 16, 1952. She had had no obstetric care during this pregnancy. She gave a history of having had two previous cesarean sections for cephalopelvic disproportion. Because of marked weight gain, (52 pounds) and the fact that the patient thought she was approximately at term, she sought medical care at our hospital. Physical examination revealed the uterus to be enlarged to 38 cm. above the symphysis pubis. The fetus was in occiput right anterior presentation, with normal fetal heart tones. There was no pedal edema. Laboratory findings revealed a specific gravity of 1.013 in the urine, which also contained one plus proteinuria. The hemoglobin was 12.2 Gm.; leukocytes 15,400; the blood urea nitrogen 11.5 mg per cent; CO_2 19 mEq/l. Pelvic measurements revealed a generally contracted pelvis.

Because of the excessive weight gain and albuminuria, the patient was placed on our toxemia regime including ammonium chloride. On the evening of January 19, she went into active labor. She was taken to surgery and the lower portion of the old uterine scar was found to have ruptured. The scar was opened sufficiently to deliver without forceps a living 9 pound 13 ounce infant. Because the bleeding from the ragged edges of the ruptured uterus could not be controlled readily and because it was thought that repair might jeopardize the patient's life in future pregnancies, a hysterectomy was performed. The patient received 1000 cc. of blood during surgery to cover an estimated blood loss of approximately 1100 cc. She was returned to the ward in good condition, with the blood pressure being maintained at 108/60. Continuous intravenous fluids were maintained throughout the night. At 7:30 a.m. on the day after surgery, an additional liter of intravenous 5 per cent glucose in distilled water was started. Forty-five minutes later a marked and severe shaking chill was noted. It was also then noticed that the solution in this third intravenous bottle, which

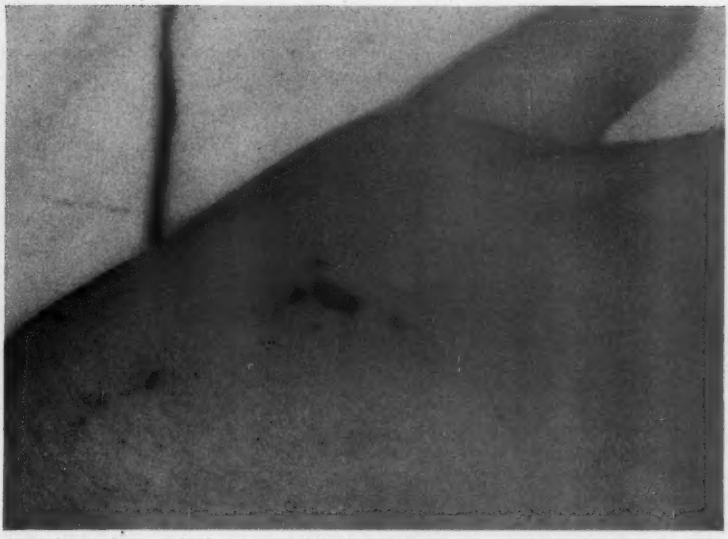

FIG. 2. Area of necrosis dorsum of right hand which occurred simultaneously with nor-epinephrine ulcers upon the leg of patient number 2.

the patient was receiving, was markedly cloudy, the patient having received 150 cc. from this bottle. At this time, her temperature was 103.4°, the pulse 180, and the blood pressure 70/63. Within 20 minutes the blood pressure dropped to 60/40. The urinary output up to this time had been 3000 cc. During the next 3 hours the urinary output dropped to 15 cc. and was dark brown in color and viscid in consistency. That evening her condition became progressively worse, with the blood pressure dropping to 40/0, the pulse still 180. In addition to other therapy given to combat this shock-like picture, the patient was started on intravenous nor-epinephrine (8 mg. or 4 cc. per liter, at the rate of 40 drops per minute) through a cut-down polyethylene tubing inserted just above the internal malleolus and fixed into the small saphenous vein. Approximately 14 hours after the infusion of nor-epinephrine had been started, a 3 by 3 cm. vesicle was noted over the medial surface of the left gastrocnemius muscle approximately 12 cm. beyond the termination of the polyethylene tubing. This at first was considered to be a superficial burn due to the application of a hot water bottle, but careful investigation revealed that no hot water bottle or other heat had ever been applied. Concomitant with the bleb formation on the left leg, a small area of discoloration was noted on the dorsum of the right hand (figure 2). This discoloration was due to superficial necrosis of skin which did not proceed to ulceration. No previous trauma to this area was recorded. At this time the patient's BUN was 37.8 mg. per cent but later rose to 70 mg. per cent, the CO_2 was 14 mEq/l, and the serum sodium had dropped from 141 to 124 mEq/l. Blood cultures were taken, as well as urine cultures. A culture of the contaminated intravenous fluid was also done and after careful analysis was reported 3 weeks later as being contaminated with aerobacter achromogen and with monilia.

Throughout the patient's course of the next 48 hours, the blood pressure was at critically low levels, and responded only as long as nor-epinephrine was continued. The urinary output was suppressed practically completely but it, too, was stimulated as long as nor-epinephrine was administered. As soon as nor-epinephrine was discontinued blood pressure and urinary output dropped to imperceptible levels. During the course of the patient's hospitalization she showed evidence of gastrointestinal ulceration by the development of hematemesis which developed during the clinical course, hematuria, melena, as well as the hemor-

rhage into the vesiculation which developed in the left leg. The critically low levels of electrolytes were replaced accordingly.

The ulceration in the left leg became more pronounced and was treated conservatively. It was extremely slow in granulating and required approximately 2½ months before it healed.

Case 3. Mrs. L. N., a 24 year old Para II, Gravida III, white woman, who had never had any prenatal care during this pregnancy, was admitted to the hospital at 2:00 a.m. on Feb. 5, 1952. Shortly after admittance to our hospital, the patient spontaneously aborted a 20 cm. fetus and an intact placenta. The total blood loss was approximately 50 cc. It was later learned during the course of this hospitalization for the treatment of this complicated pregnancy that the patient had been frequently and intermittently inserting a piece of old coil spring into the cervix to evoke abortion.

General physical examination revealed a well-developed, well-nourished adult white woman not appearing acutely ill, but with a moderate complaint of discomfort and in a very pronounced state of anxiety. Her blood pressure was 90/60, the pulse 100, temperature 101°, respirations 24. The patient was cold and clammy. On admittance the patient was catheterized but no urine was obtainable. Five hours later, the first obtainable catheterized specimen consisted of 20 cc. of dark brown urine. However, during the first 24 hours of hospitalization, the total urinary output was 30 cc. Twelve hours after admittance, she had the onset of severe headache, nausea and vomiting, and the passage of involuntary stools. A spinal tap was performed and revealed normal hydrodynamics and cytology. Fifteen hours after admittance, the patient complained of generalized malaise, paresthesias of the extremities, aching in the masseters, inability to open the mouth and delirium. Upon examining the patient at this time, she was noted to be semicomatose with hyperactive reflexes and pronounced trismus. The blood pressure varied between 70/50 and 82/58.

Laboratory data: Hemoglobin 11.5 Gm., hematocrit 41 per cent, leukocytes 24,000 with 92 per cent neutrophiles, urinalysis revealed 60 mg. of protein with 12 red blood cells per high power field and 4 white blood cells per high power field. Klein test was negative. Cervical smear showed many pus cells and gram negative rods. Three successive blood cultures drawn on the date of admittance revealed no growth at the end of 10 days.

FIG. 3. Early blebs which formed prior to ulceration of the leg in patient number 3. Upper bleb was 20 cm. above tip of nor-epinephrine injecting cannula.

With a tentative diagnosis of tetanus septicemia, the patient was given an initial dose of 1500 units of tetanus antitoxin, followed by two therapeutic doses of 40,000 units each, providing a total dosage of 81,500 units of tetanus antitoxin. She was also started on 2 million units daily of penicillin. She was placed on intravenous fluids. Because one could not be certain of blood loss, as evidenced by the shock-like picture, a pulse of 112, and a hematocrit drop from 41 per cent to 35.5 per cent, 500 cc. of blood was administered. Just prior to its administration the leukocytes were 39,700. In addition to the 5 per cent glucose, intravenous nor-epinephrine was begun through a polyethylene catheter in an incision similar to the one made in the patient in Case 2, in concentration of 4 cc. or 8 mg. per liter. The nor-epinephrine produced an elevation of blood pressure to 100/80 but there was no excretion of urine. Nor-epinephrine was continued in the second 1,000 cc. of 5 per cent glucose in water. Six hours later the urinary output had reached 115 cc. It then gradually increased over the next 24 hours. Each time that the nor-epinephrine was discontinued blood pressure and urine levels simultaneously decreased to extremely low levels.

Forty-eight hours after admittance the patient had responded fairly satisfactorily, but began to complain of generalized right leg tenderness with cramping pains in the lower right leg. Examination of the extremity revealed two areas approximately 3 cm. in diameter with an irregular border and a deeply erythematous base. One of these areas was located just about at the tip of the catheter, while the other was present just below the knee joint (at least 20 cm. above the termination of the cannulation). Six hours after these areas were noted, the borders of the lesions assumed a violaceous hue: the lesions were entirely flat but later became bullous (figure 3). There was mild edema and no suggestion of thrombophlebitis. On the following morning, February 8, there was definite and marked swelling of the right leg with generalized tenderness, the circumference of the extremity being 6 cm. larger than that of the left extremity. The lesion became deeper and continued to excavate becoming a deep ulcer and extending through all the superficial tissues down to the deep muscles of the calf. The lesion was treated conservatively, granulated in very slowly, and ultimately required a split thickness graft which healed without event.

Case 4. Mrs E. W., a 46 year old white woman, Para I, Gravida II, Aborta I, was first seen in the Emergency Room of our hospital at 5:00 a.m., June 14, 1954, and admitted from the Emergency Room to the Surgery Service with chief complaints of abdominal pain and diarrhea of 24 hour duration. Over a period of 3 days prior to admission a generalized abdominal pain had localized to the lower abdomen, primarily on the right.

Physical examination revealed a well developed, thin, white female patient, appearing acutely ill and complaining of generalized abdominal pain. On admission the pulse was 90, the temperature was 103°, the respirations were 18 and the blood pressure was 120/80. The abdomen revealed rigidity and tenderness over both lower quadrants. There was rebound tenderness over the entire lower abdomen. The bowel peristalsis was absent to auscultation.

Pelvic examination revealed evidence of marked pelvic tenderness in addition to the pronounced tenderness over McBurney's point. The cervix was clean and parous, with a small anterior eversion. There was a moderate amount of clear mucoid discharge in the vagina. The cervix was markedly tender to motion. There were no palpable adnexal masses, but marked tenderness was present bilaterally.

Urinalysis of a catheterized specimen revealed 30 to 40 red blood cells, but with no other elements. There was insufficient quantity to determine specific gravity. Hematocrit was 40 per cent, leukocytes 11,300 with 88 per cent neutrophils. Cervical smear revealed gram positive cocci, occasional yeast cells, but no gonococci.

Because of a blood pressure of 80/55, the tachycardia, and a picture of collapse, it was thought that her shock-like state might well be due to intraperitoneal bleeding. Neosynephrine was started by intravenous drip with the blood pressure returning to normal levels. However, when the drip was discontinued, the blood pressure again fell to shock levels. Consequently, at 6:00 p.m., a laparotomy was performed revealing acute bilateral suppurative salpingitis but no evidence of hemoperitoneum or pelvic abscess. The abdomen was then closed without further procedure. The patient was returned to bed and placed on a

conservative regime for peritonitis. Vasopressor agents were discontinued at 8:30 p.m., but the blood pressure again fell progressively to 70/40. Intravenous neosynephrine was restarted with the blood pressure response to 80/60. Despite an increase in rate of administration of neosynephrine, the blood pressure continued to drop further to a point where it was no longer obtainable. It was then decided to administer nor-epinephrine. The right small saphenous vein was exposed and an inlying polyethylene tube was introduced at the ankle. Intravenous nor-epinephrine was then begun in concentration of 4 mg. per liter. After 30 minutes of the infusion, the blood pressure rose to 110/70 with an associated improvement of the quality of the peripheral pulse. The pressure remained at these normal levels throughout the night. However, on the following morning, it was noted that upon discontinuing the nor-epinephrine the blood pressure again fell to shock levels; thus a continuous intravenous infusion of 1,000 cc. of 5 per cent dextrose in water containing 6 mg of nor-epinephrine was maintained. Because the patient was in positive water balance, the rate of administration of fluid was reduced. Diuresis did not occur until 72 hours after admittance with the return of electrolytes to normal from very markedly low levels.

On the fourth day after admittance, small blebs were noted over the medial surface of the right thigh. It was anticipated that these would break down but no ulceration occurred. At the same time the patient developed mild superficial hemorrhagic bilateral conjuctivitis which never went on to ulceration. The blood pressure and pulse, after being maintained at normal levels with nor-epinephrine for 72 hours, then remained consistently normal after discontinuing the drug. Her postoperative period remained entirely normal throughout the remainder of her hospitalization and she was discharged on her thirteenth postoperative day. Follow-up examination revealed no latent sequelae.

Case 5. Mr. P. W. S., a 43 year old white man was admitted to the hospital with mitral stenosis with auricular fibrillation secondary to rheumatic heart disease. A mitral commissurotomy was performed on June 18, 1952. During the 2 hour operative procedure the blood pressure was unstable. Nor-epinephrine, 4 mg./1000 cc. of 5 per cent glucose in distilled water was begun in the right ankle through a no. 15 gauge needle. Approximately 200 cc. of this solution was given intermittently during the operation and immediately postoperatively. The following day, June 19, at 2:00 p.m., a bluish discoloration with adjacent blanching hyperemia was noted over the site of the infusion. Bedside mecholyl iontophoresis was carried out the following morning. A blister over the area was observed in the afternoon. A dry sterile dressing was applied. A watery drainage was noted on the fifth postoperative day. The ankle, although uncomfortable, did not prevent ambulation by the seventh day and discharge on the twelfth postoperative day. The discoloration gradually resolved without loss of skin.

Case 6. Mr. W. S. suffered from severe mitral stenosis and auricular fibrillation secondary to rheumatic heart disease. On June 25, 1952 mitral commissurotomy was carried out uneventfully except for a blood pressure drop. The pressure was supported by small amounts of nor-epinephrine (Levophed) solution containing 4 mg./1000 cc. of 5 per cent glucose in distilled water. This was administered intermittently during surgery through a polyethylene catheter placed prior to surgery by means of a small cut down in the right ankle. The operating time was 2 hours and 10 minutes. The nor-epinephrine solution was begun 50 minutes after the beginning of surgery and was discontinued at the conclusion of the operation. In the afternoon of surgery a well demarcated bluish discoloration with peripheral hyperemia was noted at the infusion site. Early the following morning this area was treated by bedside mecholyl iontophoresis. Five hours after treatment only slight discoloration remained; there were three small areas of blister. The following day, June 27, a second iontophoresis was given with a good reaction noted. The blister area drained subsequently, with a gradual resolution of the area without loss of skin. Although the patient had some discomfort in this area, it did not prevent him from walking up and down the corridor without difficulty.

Case 7. Mrs. M. W., a 76 year old Negro woman, was admitted to the hospital for a large bowel obstruction for which a cecostomy was performed on August 10. Her convalescence

was uneventful until August 24, when suddenly the patient became cold, clammy, pulseless and without an obtainable blood pressure. A pulmonary embolus was suspected. A cut down was immediately performed and a nor-epinephrine (Levophed) solution 4 mg./1000 cc. was started at 11:00 p.m. This was continued slowly at 30 drops per minute. The blood pressure response was good and the solution was discontinued the following morning. A bluish discoloration at the infusion site extending up along the course of the greater saphenous was noted. A dry sterile dressing was applied. Necrosis became evident and the area became mummified. On September 26, a palliative segmental sigmoid colectomy for carcinoma was performed. Hepatic metastases were present. On October 15, the leg was debrided. On Oct. 27, 1952, the cecostomy was closed after the leg ulcer was narrowed by suture. One month later the area was still healing. It was obvious that it required grafting. However, in view of the poor general condition of the patient, it was treated conservatively. At the last out patient visit, the area was well healed; the scar measured 10 cm by 2.7 cm.

Case 8. Mr. K. V. a 74 year old white man, was admitted to the hospital on several occasions for cholelithiasis and acute recurring pancreatitis. He had, in addition, severe arteriosclerotic heart disease with auricular fibrillation which was at times difficult to control. On his first admission (April 3, 1953) a cholecystostomy was performed complicated by a severe blood pressure fall. Intravenous nor-epinephrine solution 4 mg./1000 cc. was given by drip through a needle in the right medial ankle. A bluish discoloration was noted at the site of injection and a dry sterile dressing applied. He was ambulated on the third postoperative day. The well demarcated bluish area slowly became black and mummified. This area was debrided on April 25, and grafted. An additional graft was applied on May 14, 1953. The largest diameter of the grafted site after healing was 6 cm. Two additional subsequent admissions occurred for recurring pancreatitis. After conservative treatment resulted in some betterment, eventual cholecystectomy was performed.

Case 9. Mr. J. R., a 53 year old white man, known chronic alcohol addict, was admitted to the hospital in October of 1954 for severe upper gastrointestinal bleeding. The patient's course was complicated by an episode of hypochloremic alkalosis secondary to preadmission vomiting. The blood pressure could not be obtained on admission, and nor-epinephrine solution 4 mg./1000 cc. was begun through a polyethylene tube inserted by means of a cut-down into a left ankle vein. An unknown amount of nor-epinephrine was given; however, 48 hours after admission there was noted to be an "annular ischemic ring just below the left knee". The cut-down was discontinued at that time. The ischemic area on the left upper leg progressed to necrosis and ulceration (figure 4). Specific local therapy was not given in an early phase. A skin graft was applied later to the granulating wound; however, healing was substandard and when the patient was last seen in mid January of 1955, the wound was not completely healed.

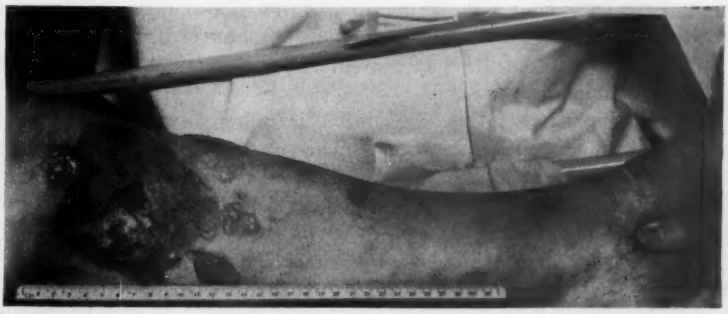

FIG. 4. Massive necrosis of tissue following use of nor-epinephrine solution in patient number 9.

Therapy is most effective when nor-epinephrine solution tissue infiltration is recognized early. The early local application of heat is condemned because cellular metabolic requirements are increased without concomitant vasodilatation. Injection of hyaluronidase and procaine 1 to 2 per cent into the affected area has aborted the persistent ischemia in some cases before permanent damage has occurred. We have had exceptionally good success with the use of mecholyl iontophoresis. This therapy was applied early after recognition of the existing problem in several of the reported cases, and points as much to the importance of early diagnosis as to specific methods of therapy. Close and Kory[4] recently have demonstrated the efficacy of infiltration of Regitine or Priscoline in saline solutions into the site of nor-epinephrine extravasation to prevent tissue breakdown. Hyaluronidase alone was ineffective according to these latter workers.

EXPERIMENTAL STUDY

Even though the clinical occurrence of tissue necrosis occurred following the administration of nor-epinephrine, many other conditions were also simultaneously present. Thus it was thought desirable to explore the possibilities of causation of the tissue ulceration with each of these conditions operating alone. The circumstances which had to be considered in the causation of the ulceration included the possible intradermal escape of the drug, the local pooling of nor-epinephrine solution, the use of polyethylene cannulation, the presence of shock, the presence of blood loss prior to shock, tissue tension, as well as the influence of extravasation of the nor-epinephrine as a complication of intravenous administration. In order to study these various influences dogs were used as experimental subjects to study nor-epinephrine in the production of ulcer and other tissue changes.

METHODS

Eight healthy mongrel dogs weighing from 8.5 to 15 Kg. were used in these experiments. Intravenous sodium pentobarbital was used for anesthesia. Aseptic surgical precautions were maintained throughout all procedures.

The concentration of nor-epinephrine injected was constant throughout all these experiments. Four mg. of nor-epinephrine (Levophed) were diluted in 1000 cc. of 5 per cent glucose in water or in one liter of 0.85 per cent sodium chloride. Thus a concentration of nor-epinephrine equal to 4 micrograms per cc. of injected fluid was obtained.

The nor-epinephrine solution was injected intradermally, subcutaneously and intravenously under various conditions. The sites of injection were closely observed for a minimum period of 3 weeks. If no ulceration developed at the conclusion of this or a longer period of time, some of the animals were reinjected at different body sites.

The majority of the subcutaneous injections of nor-epinephrine solution were small in quantity (5–20 cc.) thus placing the tissues under no tension. However tension of various subcutaneous tissues was purposely produced in 6 experimental instances. The injection of 40–70 cc. of the standard solution subcuta-

neously under the skin of the trunk and of 20 cc. into the restricted subcutaneous space of the ears produced the desired degree of tissue tension.

In order to simulate the administration of nor-epinephrine to patients, all intravenous solution was given through a polyethylene cannula. Each cannula was threaded into each vein to a measured distance through small skin incisions. Careful technic was employed to prevent extravasation of the nor-epinephrine solution in all but one instance. In Dog 2029 a 23 gauge needle hole was purposely made in the vein near the tip of the cannula.

Acute shock was produced in four instances by rapid removal of blood from one of the femoral arteries. Accurate blood pressure changes during the shock experiments were obtained by the use of a Sanborn multichannel polyviso recorder. Intravenous nor-epinephrine was then administered for varying periods in attempts to control the vascular collapse obtained. After responding to the nor-epinephrine solution, the dogs were then transfused with the preserved phlebotomized blood. These animals were then closely observed for evidence of possible skin ulceration.

RESULTS

These experiments were divided into three groups. The animals in Group I received the standard nor-epinephrine solution either by simple subcutaneous or by intradermal injection. In most instances, the solution was administered without tension of the subcutaneous tissues. Among the Group II dogs a state of shock from acute blood loss was induced. While in this state of acute vaso-motor collapse, a standard solution of nor-epinephrine was injected intravenously. To the Group III dogs simple intravenous injections of varying amounts of nor-epinephrine solution were administered with particular care being taken to prevent or obtain extravasation of the solution.

Group I (table 1): Seventeen separate subcutaneous or intradermal injections of nor-epinephrine solution (4 micrograms/cc.) were administered. Ten of these injections were of small quantity and tissue tension was not present. A small skin ulceration (1 by .5 cm.) developed at the site of injection in one dog, Y 65, on the seventh day. This ulcer was excised in toto soon after its appearance and was subjected to gross and microscopic study. The most striking gross feature of this and all the other excised specimens was the marked edema present in the subcutaneous tissue surrounding each ulcer. Microscopically this was characterized by the swollen and separated collagen fibers found in the ulcer base from Dog Y 65, and is well demonstrated in figure 5.

Seven injections of the standard solution under tension were given with the production of two ulcers. Extensive breakdown of the skin and the subcutaneous tissues followed the injection of 70 cc. of nor-epinephrine solution into the right shoulder region of Dog 1900. This ulceration occurred not only at the site of injection, but also in the right axilla and right chest (figure 6). This would seem to indicate that a portion of the "poorly absorbed" fluid gravitated to the more dependent portion of the adjacent trunk, and there caused additional necrosis of tissue. A superficial ulceration developed on the ear of Dog 1903 occurring

GROUP I

SIMPLE SUBCUTANEOUS INJECTION OF NOR-EPINEPHRINE

Dog	CC. Nor-epinephrine solution injected	Site of injection	Tissue Tension	Result
Y 63	10	Left shoulder	No	Negative
	10	Left foreleg	No	Negative
	5	Left iliac crest	No	Negative
	5	Left hindleg	No	Negative
	1.4 (Intradermal)	Right foreleg	Yes	Negative
Y 65	10	Right shoulder	No	1.0 x 0.5 cm. ulceration
	10	Right foreleg	No	Negative
Y 66	10	Left shoulder	No	Negative
	5	Left iliac crest	No	Negative
	10	Left thigh	No	Negative
	5	Right thigh	No	Negative
1900*	20	Right ear	Yes	Negative
	40	Right thigh	Yes	Negative
	70	Right shoulder	Yes	7 x 9 cm. deep ulceration
1903**	60	Left foreleg	Yes	Negative
	20	Left ear	Yes	Negative
	20	Right ear	Yes	Superficial ulceration

* See Figure 6
** See Figure 7

TABLE 1

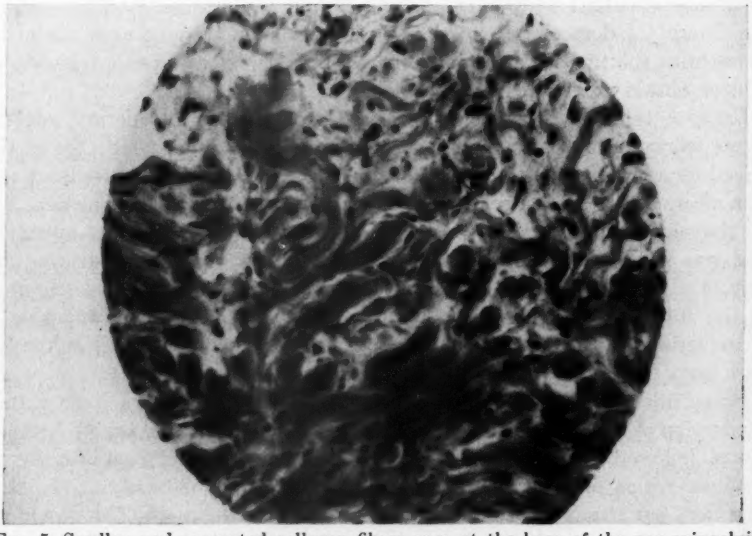

FIG. 5. Swollen and separated collagen fibers seen at the base of the nor-epinephrine ulcer excised from Dog Y65 (×220). This is indicative of the marked subcutaneous edema found in all the nor-epinephrine ulcers.

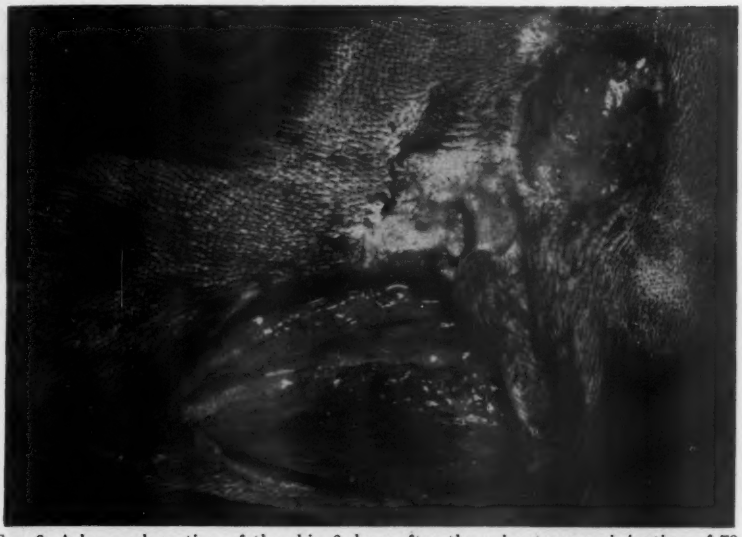

FIG. 6. A large ulceration of the skin 6 days after the subcutaneous injection of 70 cc. nor-epinephrine solution (4 micrograms nor-epinephrine/cc.) into the right shoulder region of Dog 1900.

48 hours after apparent failure of absorption of the injected fluid. The ear maintained a thickened, boggy consistency during this 48 hour period. The formation of small skin vesicles occurring approximately 48 hours after injection was soon followed by complete breakdown of the superficial epithelium (figure 7).

Group II (table 2): Since the clinical situations where nor-epinephrine is indicated are often associated with acute hemorrhage and shock, similar circumstances were produced in dogs and then treated. In the clinical patient suffering from acute blood loss, it was thought that shock and the diminished blood volume probably were prominent factors contributing to tissue damage. With the attendant vasoconstrictive effect of nor-epinephrine, the possibility of augmented tissue necrosis loomed high. Thus, acute peripheral vascular collapse was produced in 4 instances by the rapid withdrawal of blood. Subsequent control of the shock-like state was almost completely accomplished by the intravenous administration of varying quantities of the nor-epinephrine solution. However, complete control of shock was maintained only after the restoration of normal blood volume. The nor-epinephrine was injected into the foreleg vein of the dogs using a polyethylene cannula, just as had been used among patients who had developed nor-epinephrine ulceration. In these experiments, no extravasation of the nor-epinephrine solution occurred. No particular pallor, cyanosis, decrease in skin temperature, or venospasm was detected at the site of entrance of the cannula, at or near the cannula tip nor at any location in the involved extremity during injection. On subsequent examinations, no ulcerations were observed.

FIG. 7. Superficial skin ulceration right ear of Dog 1903, 72 hours after subcutaneous injection of 20 cc. nor-epinephrine solution.

GROUP II

INTRAVENOUS INJECTION OF NOR-EPINEPHRINE DURING SHOCK

Dog	Site of vein cannulated	CC. Nor-epinephrine solution injected	Result
Y 63	Left foreleg	30	Rapid blood pressure response No subsequent ulceration
Y 64	Left foreleg	200	No ulceration developed at site of injection or on the distal extremities
Repeated at later date	Right foreleg	150	No ulceration developed at site of injection or on the distal extremities
Y 66	Left foreleg	150	No subsequent ulceration

TABLE 2

The rapid blood pressure response to nor-epinephrine was readily noted on the Sanborn polyviso recording apparatus. The average time of blood pressure response from an imperceptible level to a significant rise was only 12 seconds. The blood pressure response could be rapidly titrated by the simple expedient of increasing or decreasing the rate of flow of nor-epinephrine, a response so characteristically noted among patients in shock.

Group III (table 3): In only one instance (Dog 2029) did tissue necrosis or skin ulceration develop following the simple intravenous administration of nor-epinephrine (figure 8). During the continuous intravenous administration of

800 cc. of the standard solution over a 6 hour period, an estimated 30 cc. extravasated into the subcutaneous tissues. The sequence of events which followed was generally typical of all the ulcers regardless of the methods used to produce them. During the first 24 hours after subcutaneous infiltration of the fluid, either by simple injection or extravasation from a cannulated vein, there appeared to be a complete lack of absorption of the fluid. The overlying skin appeared blanched, edematous and was distinctly colder than the surrounding skin.

GROUP III

SIMPLE INTRAVENOUS INJECTION OF NOR-EPINEPHRINE
(WITH AND WITHOUT FLUID EXTRAVASATION)

Dog	Site of vein cannulated	CC. Nor-epinephrine solution injected	Extravasation of Nor-epinephrine solution	Result
Y 64	Right foreleg	25	No	Negative
Y 66	Right foreleg	150	No	Negative
2023	Left foreleg	800	No	Negative
2029*	Left foreleg	800	Yes (Estimated 30cc.)	Large 6x7cm. ulceration at site of extravasation

* See Figure 8

TABLE 3

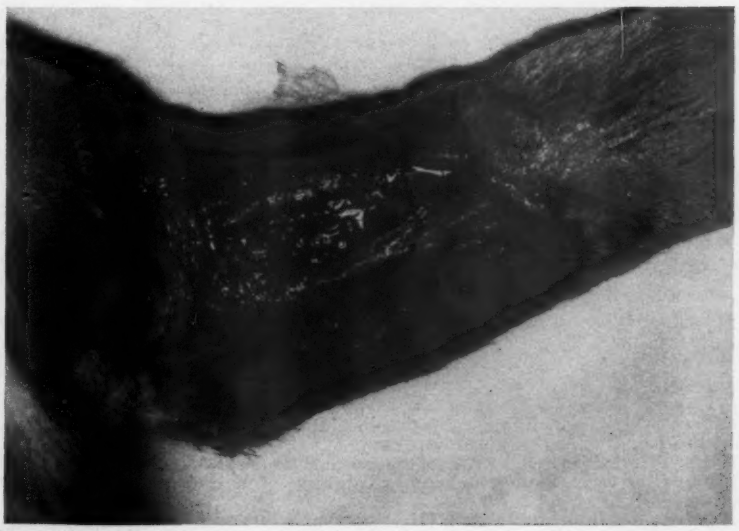

FIG. 8. Nor-epinephrine ulcer left foreleg of Dog 2029, 7 days after extravasation of an estimated 30 cc. of nor-epinephrine solution.

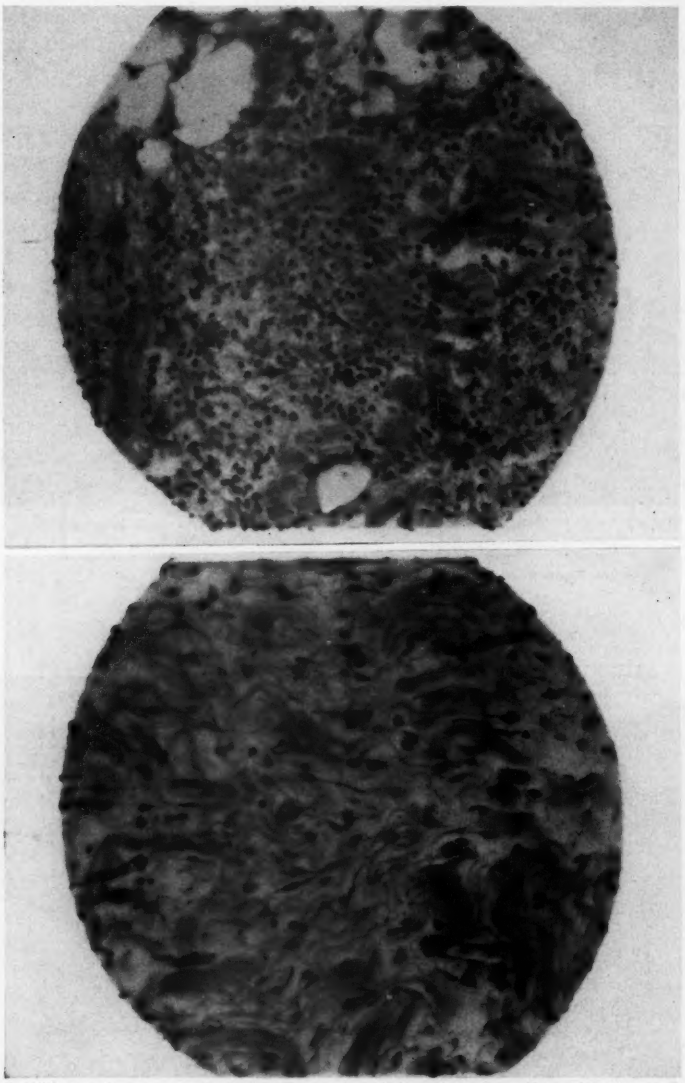

FIG. 9a. Photomicrograph (×220) of nor-epinephrine ulcer from Dog 2029 showing clearly the marked polymorphonuclear cell response.

FIG. 9b. Photomicrograph (×220) of the base of the nor-epinephrine ulcer from Dog Y65 showing the early fibroblastic activity indicative of an early attempt of healing in these lesions.

Within 48 hours the skin was still blanched and the tissues still boggy. Small vesicles had appeared on the skin surface. These readily broke down with subsequent exudation of a serosanguinous fluid. Superficial necrosis became apparent 72 hours after injection with complete breakdown of the affected area on about the sixth day. One of the most striking features of these ulcers was the marked polymorphonuclear cell response to the injury (figure 9a). Fibroblastic proliferation was evident at the base of each ulcer indicative of an early attempt at repair in these lesions (figure 9b). In general the microscopic picture was that of ulceration of the skin surface, necrosis, and an acute inflammatory response.

DISCUSSION

The production of large necrotic ulcerations near the site of injection following intravenous use of nor-epinephrine solution is not an uncommon complication following this therapy. It has been believed that this ulceration was due to inadvertent extravasation of the solution into the tissues with subsequent vasoconstriction, ischemia, and necrosis. We were unable to experimentally produce ulceration when the solution was given only by vein. This was also the experience recently reported by Berben, Bryant, and Howard.[1] Thus a majority of ulcerations occur due to some mechanical factor such as puncture of the vein wall by a needle, allowing the solution to extravasate freely into the tissues. Close and Kory[4] noted marked spasm of the vein into which nor-epinephrine solution was being injected. These latter authors also noted an associated ischemia of the vein wall and, therefore, they postulated that this ischemia permitted diffusion of the nor-epinephrine solution into adjacent tissues.

Remote areas of necrosis or vascular damage after the use of nor-epinephrine solution is suggested by the presence of the remote degenerative ulcerative necrotic area which occurred in the hand of one of our patients and the conjunctival changes which occurred in the fourth patient presented herein. We differ with most authors in that we do not believe that the effect of nor-epinephrine is necessarily limited to the site of infusion of the drug, but that the effects of the drug exhibit marked generalized vasoconstriction producing ischemic necrosis of skin and subcutaneous tissue by its profound pressor effect. Bricker[3] in 1954 stated that a patient under his surveillance developed a necrotic "nor-epinephrine" ulcer high in the upper arm spatially far removed from the site of injection. This latter observation would tend to confirm the occurrence of vascular changes in areas remote from the possible area of extravasation as noted by us. The exact mechanism whereby nonspecific sites are chosen for necrosis is not understood.

It would thus seem that the effect of nor-epinephrine, in addition to being a powerful local vasoconstrictor, exerts intensive vasospastic influences throughout the entire body, enough so in large and continued dosage to provoke severe ulcerative damage. These severe changes occur particularly where blood flow has been previously reduced by prolonged shock or by other hypotensive mechanism. In severe hypotension, the nephron is affected by persistent reduction of the glomerular filtration rate and renal blood flow. While the vasoconstrictor

influences of nor-epinephrine occur in the peripheral vessels, the increase in urinary output reflects an increase in glomerular filtration rate to approach physiologic levels in these deranged pathologic states associated with shock. While it has been shown by Smythe and associates[10] that renal plasma flow is slightly decreased following the administration of nor-epinephrine, it would seem that the filtration fraction remains essentially unchanged in order to explain the increased urine flow associated with the use of the drug.

CONCLUSIONS

A. General

1. Nor-epinephrine is a powerful vasoconstrictor agent; it is also an effective adjuvant in the therapy of acute renal failure secondary to vasomotor collapse.

2. On the basis of our experience, nor-epinephrine may be classified as a local tissue irritant, producing local tissue and vascular necrosis where the conditions of tension by and/or extravasation of nor-epinephrine prevail. It is entirely possible that increased capillary permeability due to shock contributes to such extravasation.

B. Experimental

1. Nor-epinephrine as generally administered for the control of vasomotor collapse is an extremely potent vasoconstrictor.

2. The effects of nor-epinephrine in producing ulceration could be demonstrated only locally. Under the conditions of these experiments, only local ulceration at the site of nor-epinephrine leak could be produced. Distant ulceration could not be identified.

3. Intradermal administration of nor-epinephrine did not result in local ulceration.

4. Significant ulceration appeared in two dogs when nor-epinephrine was administered subcutaneously to a point of tension.

5. Under the conditions of shock no evidence of tissue ulceration occurred in dogs following the intravenous administration of nor-epinephrine. Perhaps vasomotor influences which would increase vascular permeability were not operating in sufficient degree or sufficient duration to allow extravasation.

6. Following the simple intravenous administration of nor-epinephrine, extensive, deep tissue necrosis and ulceration occurred only at the site where extravasation of nor-epinephrine was permitted in the dog.

C. Human

1. Nor-epinephrine in concentration of 4 to 8 mg. per liter produces marked local tissue necrosis in the human.

2. Nor-epinephrine, when infused intravenously, produces marked generalized vasoconstriction with ischemic tissue necrosis at points remote from the site of infusion.

3. While nor-epinephrine is life-saving under some circumstances, its local

and remote effects should be carefully assayed in light of the strong medical-legal implications associated with its use.

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CAUSALGIA

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Following any injury to an extremity, whether accidental or surgical, infectious or noninfectious, venous or arterial, peripheral nerve or not, a bizarre symptom complex called causalgia occasionally occurs. This syndrome is characterized by an overactive sympathetic nervous system, superficial pain exceeding the expected, and hyperesthesia of the involved part.

Potts[14] first mentioned this response to trauma in the late eighteenth century. Mitchell, Morehouse, and Keen[13] are credited with the first classical description of the syndrome when, in 1864, they recorded their observations in soldiers with gunshot wounds of peripheral nerves. Weir Mitchell,[12] 8 years later, introduced the term "*causalgia*" to describe the outstanding symptom, burning pain. His original definition was "hyperesthesia of the hand or foot following an injury in the region of a peripheral nerve". The term "*causalgia*" has since been applied to many variations of the syndrome originally reported. Clinicians in different specialty groups disagree as to exactly what the entity "*causalgia*" is, and use various names for it according to the focus of attention on different manifestations. Best known among these names are acute atrophy of bone. Sudeck's atrophy, traumatic vasospasm, traumatic angiospasm, reflex nervous dystrophy, post-traumatic osteoporosis, minor causalgia, neurovasospastic phenomenon, and chronic traumatic edema.

ETIOLOGY

Many theories have been proposed as to the cause of causalgia, but none is completely satisfactory.

Mitchell,[12] in his original articles, advanced no tenable explanation. Lewis and Pickering[9] described accessory nerves (nocifensor fibers), McKenzie[5] the reflex arc, Tinel[6] blamed infection, and Leriche[8] proposed reflex vasospasm. Other authors suggested that the sympathetics functioned as an efferent pain pathway, as in visceral pain. Some clinicians adopted the theory of fibrosis about the nerve. None of these theories has been proved, since causalgia did not occur in every case.[10]

Doupe and associates[4] in 1944 advanced the most logical theory to date—a shunting of impulses from the sympathetic to the sensory fibers of the injured nerve. White and associates[18] went farther and stated that the efferent impulses from the hypothalamus travel to the sympathetics following emotional and environmental stimuli.

Certain writers hold that different noxia are responsible for the production of

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Read before the Third Latin-American Congress of the International Society of Angiology, Havana, Cuba, Nov. 10, 1956.

the syndrome in the presence of an endogenous abnormal reactive predisposition of the sympathetic nervous system.[1, 2, 15] In reviewing the histories of these patients, one can not help believing that the 5 per cent or less[7] reported as having this condition following nerve injuries must have a different anatomic and physiologic makeup from that of the other 95 per cent of the population. It appears that most of these patients tend toward periods when their sympathetic nervous systems are hyperactive, either obviously—on an emotional basis—or by clinical signs, and are therefore prone to develop causalgia following trauma. In our clinic, we term this type of individual a "*sympathetic reactor*".

Frequently, a psychogenic etiology is suggested, and many patients have been unjustly considered to be malingerers or hysterics. No constitutional psychogenic factor can be found, but sudden noises or emotional stimuli such as fear or anger do influence the pain, especially in extreme cases and in the direct nerve injury group.

SYMPTOMS

The outstanding feature of the disorder is *burning superficial pain*. The superficial character is especially emphasized to differentiate it from deep pain such as that caused by ischemic neuritis, traumatic neuritis, abscess, neuromas, bone injury, et cetera. The pain may be described as throbbing, aching, vise-like, and so forth; frequently it is difficult to obtain the classical description from the patient.

Pain may occur instantaneously, or may be delayed for weeks. Referral is distal, and chiefly occupies the palm of the hand, the fingers, toes, or plantar surface of the foot.

Hyperesthesia is the second most prominent complaint. This may be localized to a sensory nerve, but is frequently incomplete. Slight stimuli, such as drafts, bed-sheets, or examiners' hands, will induce extreme paroxysms of pain. To protect the injured part from such stimuli, patients go to extremes, sometimes holding the extremity in one position or covering it with dry or moist cloths. The cool or warm moist cloth, surprisingly, provides relief in a significant number of patients.

Increased sweating of the involved foot or hand may be very marked, and a past history regarding this tendency is frequently elicited.

Vasomotor changes vary widely. In general, these may be divided into those of vasoconstriction and those of vasodilatation. Such signs may be misleading: initial warm rubor may develop into the pale, cool, wet foot manifesting vasoconstriction—a classical picture. Arteriosclerotics most frequently show the shiny, scaling, dry foot.

Other signs may include swelling, stiffness of the joints (from disuse), and roentgenologic evidence of spotty osteoporosis.

In general, diagnosis is based on (1) history of overactive sympathetic nervous system; (2) superficial pain exceeding the expected; and (3) hyperesthesia localized to a sensory nerve.

CLINICAL MATERIAL

Fifty patients (34 males and 16 females) with superficial pain exceeding the expected in an extremity, and with at least one of the aforementioned symptoms, form the basis of this report. No patient with obvious partial severance of a peripheral nerve has been listed. All were observed on the Peripheral Vascular Disease service of Colorado General or Denver General Hospital, and all are from the indigent or low-income group. Ages varied from 14 to 77 years, with an average of 53.

Thirty-two of the 50 patients were admitted for study with incorrect diagnoses (table I). Only 18 had been correctly diagnosed when first seen. In addition, 2 patients presented medico-legal problems, and 2 were industrial compensation cases.

All patients were seen initially by both an internist and a surgeon, and careful histories were recorded. No related family history was obtained from any patient.

Thirty-six presented history of previous sympathetic hyperactivity: 22 had peripheral vascular disease, manifesting arterial insufficiency of various degrees before the onset of the causalgia; 3 of these had histories of Raynaud's phenomenon. A typical example is a 50 year old man who had suffered a traumatic amputation of the distal phalanx of the finger in 1920; this finger thereafter became uncomfortable when exposed to cold, and color changes and pain in the digit caused the patient to discontinue use of a pneumatic hammer in 1942. In 1953, he dropped a 30 pound stone on his right foot and was hospitalized for 3 months because of swelling in the foot plus superficial burning pain and marked hyperesthesia of the third toe. Such histories are elicited frequently enough for the examiner to suspect that these patients are predisposed to develop causalgia following suitable stimulus.

Initiating causes have been classified into three groups: trauma, circulatory, and infection (table II). Twenty-four patients had histories of *trauma*, including 6 fractures (4 of the tibia and/or fibula, 1 calcaneus, and 1 femur). The leg and

TABLE I

Diagnosis before treatment

(50 Patients)

Incorrect		32
	Ischemic neuritis	12
	Neuroma	5
	Venous insufficiency	4
	Arthritis	3
	Psychoneurosis	2
	Thrombophlebitis	2
	Plantar fascitis	1
	Syringomyelia	1
	Poliomyelitis	1
	Gout	1
Correct		18

TABLE II

Initiating cause of causalgia syndrome

(50 Patients)

Trauma:	
Fracture	6
Frostbite	6
Phantom limb	4
Burn to foot	3
Amputation of finger	2
Blunt injury to extremity	2
Sprained ankle	1
Total	24
Circulatory:	
Arterial:	
Arteriosclerosis obliterans	12
Thromboangiitis obliterans	2
Embolus, popliteal	2
Accidental ligation, femoral	1
Venous:	
Thrombophlebitis	3
Total	20
Infection:	
Toe (with arterial insufficiency)	4
Poliomyelitis	1
Fracture with osteomyelitis	1
Total	6

foot fractures all showed roentgenologic evidence of osteoporosis and had been diagnosed as Sudeck's atrophy. Some developed causalgic pain immediately; in others onset was delayed for weeks.

Six patients with first to third degree frostbite developed their pain almost immediately after the cold trauma. Our youngest patient, 14 years old, seen with infection 12 days after exposure, was so heavily sedated that addiction was feared.

Phantom limbs were considered to be causalgia only when pain was present. Not one of these patients had deep stump pain; all described the pain as being in the phantom foot. Two of them put warm applications on the remaining foot when it felt cold, but complained that they were unable to do the same to the phantom. One patient who was a medico-legal problem suffered a burn to the foot while undergoing a bunionectomy. This patient had a psychiatric history, which naturally was a factor in the incorrect diagnosis, but there was also excellent evidence of past sympathetic hyperactivity. Conceivably, the burn may have been only an additional factor, although Fowler and Moser[5] list 3 bunionectomies among their cases.

Of the 2 amputated fingers, 1 was accidental and 1 elective. The accidental amputation was reamputated twice, but residual pain persisted. The higher

amputation was done at the patient's insistence—an industrial compensation case. This patient also had hyperesthesia and pain at two other sites—an abdominal incision and a scar of the opposite hand. The elective amputation was done in a patient with scleroderma, in whom excision of a calcaneus cutis nodule of the finger resulted in gangrene of the terminal phalanx.

Two cases were from blunt injuries, one being the patient previously mentioned as having dropped a 30 pound stone on his foot, and the second being a patient who developed pain in both knee and foot following trauma to the knee.

The one case listed as a sprained ankle showed osteoporosis along with the classical signs of causalgia.

Arterial insufficiency was the predominant *circulatory* cause. These patients had developed either sudden or progressive femoral or popliteal artery thrombosis. Ten were listed as having ischemic neuritis, and amputation was under consideration in spite of a functionally viable foot. Two patients with thromboangiitis obliterans had similar stories. The 2 popliteal emboli were in young females with good arterial circulation before the occurrence of the emboli.

Our most difficult case was a 34 year old woman whose femoral artery had been accidentally ligated during varicose vein surgery elsewhere. When she was seen in our clinic 8 months later, she was on crutches and was sensitive even to a draft on the foot. She had developed a marked equinus deformity.

All 3 patients with venous conditions initiating causalgia had deep-vein thromboses and marked evidence of being sympathetic reactors even before development of thrombophlebitis. One was an addict before the onset of causalgia.

The *infectious* group were 6 in number, 4 with infection on feet with decreased arterial supply; 3 involved the great toe and 1 the fifth toe. Amputations were being strongly considered in all patients.

One patient had had poliomyelitis 4 years previously, her right leg and left arm presenting edema, coldness, hyperhidrosis, hyperesthesia, pain, and cyanosis. These complaints delayed physiotherapy, and the patient had refused to wear a brace to her leg.

The last case listed was a fractured tibia with osteomyelitis; causalgia did not develop until infection was present.

One or both lower extremities were involved in 45 patients, all the frostbite cases being bilateral. Upper extremities were affected in 5 patients, and 1 had causalgia in an upper extremity plus a lower one.

TREATMENT AND RESULTS

Treatment demands prompt diagnosis and early institution of specific therapy. Delay results in fixed patterns of pain, with dysfunction and atrophy of the part. The 3 patients in this series who received poorest results from therapy had the longest delay of diagnosis and treatment—6 to 20 years.

Initially, the nature of the disease, its physiologic basis, and the possibility of relief from therapy are explained to the patient, in an attempt to obtain his cooperation.

One of the vasodilators (sympatholytic drugs), preferably Priscoline 10 mg. or Hydergine .3 mg., is then given intra-arterially. In the 38 patients in whom either of these drugs was used, complete or temporary relief was obtained, thereby establishing the diagnosis (table III). The majority received Priscoline; 3 obtained complete relief with this drug, and 1 had similar results with the use of Hydergine. Of 29 patients who received oral Priscoline 50 mg. three times daily, 3 had complete subsidence of pain within a week. Two of these had phantom limbs with pain, and 1 had poliomyelitis involving both a lower and an upper extremity. However, 2 years later the poliomyelitis patient developed thrombophlebitis of the lower extremity, which necessitated a sympathectomy, since the pain returned. The hand remained free of symptoms.

The third step has been procaine sympathetic block, with infiltration of the stellate ganglion for the upper extremity or the lumbar sympathetic chain for the lower extremity. Every patient obtained at least temporary relief from this procedure, and 3 received permanent relief. In 4 patients it was necessary to block T-11 and T-12 in addition to the lumbar chain.

In any patient in whom pain disappeared for 4 hours or more, repeated sympathetic blocks were justified at later dates. Two patients refusing surgical therapy have needed an occasional sympathetic block over a period of at least 2 years.

Surgical ganglionectomy is the last resort. For the lower extremities, it may be necessary to go as high as T-11, as in the procaine sympathetic blocks. Two of our patients needed higher sympathectomy, since they had only partial relief from excision of L-2 through L-4 ganglia. Two additional patients in the poor-result group following sympathectomy have since had higher paravertebral procaine blocks with temporary relief, and repeat sympathectomy is contemplated. Both of these patients had complete relief for 3 years following their initial sympathectomies, but recent return of pain and sweating indicates that higher sympathetic denervation is necessary for relief.

Three patients required additional physiotherapy to complete their treatment.

Replacement of an injured vessel, embolectomy, or thrombectomy may be necessary. The patient whose superficial artery had been accidentally ligated did not obtain complete relief of pain until after a high lumbar sympathectomy

TABLE III

Results of therapy

(50 Patients)

	Number	Complete Relief	Good	Poor
Intra-arterial vasodilator	38	4	38	—
Oral vasodilator	29	2	18	11
Sympathetic "block"	26	9	22	4
Sympathectomy	34	28	32	2
Physiotherapy	3	—	3	—

with 80 per cent relief; neurotomy and resuture of the posterior tibial nerve with partial pain returning in 4 months; tenoplasty for equinus deformity of the foot; and replacement of the injured vessel by a freeze-dried arterial homograft.

Periarterial sympathectomy has been abandoned in our clinic, although published reports[7, 8] suggested that it may be of benefit in an occasional patient.

We have previously tried sectioning of posterior roots and intraspinal injection of alcohol in patients not listed here without success; we consider this method obsolete.

Other methods recently reported as successful have been artificial hibernation,[6] chlorpromazine,[3] hexamethonium, and dibenzyline.[7] We have not had experience with any of these methods of treatment.

Rehabilitation, including physiotherapy, is necessary to combat the patients' fear that pain will return. Atrophy from disuse may develop in patients in compensation cases as part of the mechanism to postpone going back to work. Marked osteoporosis (Sudeck's atrophy) is a good indication for physiotherapy. Complete relief for those in this group may be delayed for months.

Patients who are not relieved by adequate sympathetic denervation should not be classified as causalgia, but should be studied more carefully.

In some surgical procedures undertaken to increase the circulation to an extremity, results can be judged good on the basis of loss of causalgic-type pain. In sympathectomies for arteriosclerosis obliterans, undertaken to increase the arterial flow, we are frequently unable to detect an increase in vascular supply, yet the patient's foot is more comfortable. Similarly, an arterial graft may have thrombosed, yet the patient is subjectively improved. Arterectomy and periarterial sympathectomy alone have been known to give relief. A clearer understanding of the nature of causalgia in all its variations and a closer scrutiny of such patients will help to clarify indications for therapy and to improve our diagnostic acumen.

Our results have been most gratifying, probably because of the limited selection of patients for therapy. Every patient listed has undergone the diagnostic test of intra-arterial vasodilator or a procaine sympathetic block. Only 34 patients in the series required sympathectomy; 2 others have refused surgery and have been controlled by repeated procaine sympathetic blocks, with additional benefit from physiotherapy.

SUMMARY

Fifty cases of causalgia have been presented to emphasize the variety of initiating causes of this post-traumatic syndrome.

Diagnosis is based on the history of an overactive sympathetic nervous system, superficial pain exceeding the expected, hyperesthesia to the involved part, and permanent or temporary relief following the use of an intra-arterial vasodilator or procaine sympathetic block.

Specific treatment consists of intra-arterial and/or oral sympatholytic drugs, paravertebral sympathetic blocks, or sympathectomy, plus physiotherapy.

Recognition of the syndrome is necessary, since these patients are miserable

and fixed organic changes may take place over a period of time. Such people are frequently misunderstood and discredited because the source of the symptoms is not realized.

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SIGMOID VOLVULUS INITIATED BY ANAL FISSURE

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INTRODUCTION

Volvulus refers to axial torsion of the bowel on its mesentery resulting in partial or complete intestinal obstruction with or without gangrene and perforation. It occurs most frequently in the sigmoid colon, but may be seen less frequently in the cecum, terminal ileum, small intestine or stomach. This condition occurs three times more frequently in the male with the maximum age incidence in the middle and later decades.

The most significant predisposing factor is the anatomic configuration of the sigmoid colon flexure with the limbs of the loop closely approximated by a short mesentery with a narrow base of attachment.[4] A megacolon will frequently be complicated by volvulus on this same basis.

Many statistical studies state that volvulus accounts for 10 per cent of all cases of intestinal obstruction in this country,[1] while figures from the Balkan countries and Russia may run consistently as high as 50 per cent.[2] The greater incidence is explained in part by the greater length of the sigmoid flexure in the Russians (5 times more frequent than the German figures) and the high residue colon-loading vegetable diet peculiar to east European countries. Other predisposing factors may be chronic constipation and violent peristalsis following severe cathartics.

SURGICAL PATHOLOGY

Volvulus of the sigmoid colon is frequently recurrent producing partial intestinal obstruction. The obstipation and abdominal distention are often spontaneously relieved by the escape of flatus and the passage of a large liquid stool. In these cases the bowel is usually twisted in a clockwise fashion from 180 to 360 degrees, but the tightness of the twist is more important than the number of turns in determining the degree of occlusion of the lumen and interference with blood supply. The subacute and chronic forms have a thickened mesentery which usually protects the blood supply during torsion. However, the acute phase may produce a closed loop intestinal obstruction with a hugely distended segment containing gas and fluid within a discolored, thin walled portion of bowel. Free peritoneial fluid, gangrene and actual perforation are not uncommon.

The case herein reported is of interest because it introduces an anal fissure as the trigger mechanism of a chronic recurring sigmoid volvulus cured by fissurectomy.

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CASE REPORT

The patient, A.T., is a 61 year old negro man who was first admitted through the accident ward of the Jefferson Medical College Hospital on Jan. 24, 1954 because of crampy abdominal pain, huge abdominal distention and obstipation of 48 hours not relieved by a saline cathartic. He stated that he first noted similar episodes starting 2 years earlier, recurring every 2 to 4 months, lasting 48 to 72 hours and correcting itself by the passage of a huge foul liquid stool. On one occasion he was treated in the accident ward of another hospital by the insertion of a rectal tube with complete relief of symptoms. Significant past medical history included a laryngectomy for carcinoma with tracheostomy in 1934 with no evidence of recurrence, and syphilis adequately treated by penicillin.

Physical examination revealed tremendous tympanitic abdominal distention, especially in the epigastrium and right upper quadrant. There was no rigidity, tenderness or rebound tenderness. Intestinal movement was visible across the abdominal wall and peristalsis was hyperactive. Digital rectal examination, which was extremely painful, revealed a fibrotic and stenotic anal canal that admitted only the tip of the index finger. His temperature was was 98.4; leukocytes 14,600 per cu. mm. with 78 per cent polys. A flat plate of the abdomen, which showed a tremendous distention of the sigmoid colon that almost completely filled the abdominal cavity was interpreted as a complete large bowel obstruction.

That night he was taken to the operating room and explored under endotracheal anes thesia. There was a huge chronic volvulus of the sigmoid colon occupying the right half and upper left portion of the abdomen. The bowel wall was greatly thickened and injected, without evidence of impending gangrene. The loop of bowel was rotated 360° in a clockwise fashion. A stage resection of the sigmoid was performed with a double barrel colostomy. He had an uneventful convalescence.

Three months later the patient was readmitted for closure of the colostomy. He had been asymptomatic during this interval at home. Four days after extra-peritoneal re-establishment of bowel continuity, the patient once again developed signs of large bowel obstruction. This was thought to be due to a stenotic anastomosis, but a flat plate of the abdomen revealed distention of the rectum and lower sigmoid distal to the anastomosis. Barium demonstrated some constriction of the anastomosis but it flowed through freely to the splenic flexure. The abdominal distention responded to Miller-Abbott and rectal tube decompression, and subsequently he was discharged from the hospital.

Readmission once again became necessary 3 months later because of recurrent abdominal cramps and distention limited to the left lower quadrant. His temperature, pulse and respiration were normal with leukocytes 5,000 per cu. mm. with a normal differential count. Physical examination revealed a tensely distended lower abdomen with grossly audible borborygmi. There was no tenderness or rigidity. Rectal examination disclosed a deep, chronic, punched-out posterior anal fissure with a fibrotic base. The anal canal was stenotic and extremely tender. A small anoscope was inserted with difficulty and removal of the obturator released a tremendous sustained (30–45 seconds) passage of flatus with immediate decompression of the abdominal distention and relief of crampy pain. A 14 inch sigmoidoscope "*dropped into*" an atonic cavernous lower bowel.

The abdominal distention recurred within 24 hours and a scout film of the abdomen showed tremendous dilatation of the entire colon. Anoscopy with removal of the obturator once again immediately decompressed the bowel. Fissurectomy was advised, but the patient refused further treatment and signed his release.

He was seen in the clinic at irregular intervals complaining of abdominal distention and and recurrent anal pain with stool. Each time he was successfully decompressed via the anoscope. The chronic punched-out fissure would not heal, and the patient repeatedly refused fissurectomy.

On January 27, 1956 the patient was brought to the accident ward of Jefferson Medical College Hospital after having passed about "*a pint*" of fresh blood with clots. In the accident ward he had a spontaneous stool containing 100 cc. of fresh blood. He was taken to the

operating room, and under low spinal anesthesia, the actively bleeding deep anal fissure was excised and the advanced anal stenosis corrected by deep pectenotomy. The patient was discharged on the fifth postoperative day and his convalescence and healing were uncomplicated. To date, 10 months since correction of the anal pathology, the patient has been asymptomatic with no recurrence of abdominal distention or large bowel obstruction.

DISCUSSION

This patient's original sigmoid volvulus was caused by a combination of exciting factors. He had a laryngectomy for carcinoma with a permanent tracheostomy in 1934. These people develop a speech mechanism by eructating purposefully swallowed air.[3] Normally 80 per cent of the gas in the gastrointestinal tract has been swallowed,[4] and in the tracheotomized patient the total volume is many times greater. Aerophagia corrects itself by the passage of excessive flatus, but the painful anal fissure and accompanying sphinctor spasm would not permit adequate release of the abnormally large volume of air swallowed. This constant build up of intra-colonic pressure with distention of the narrow based limbs of the sigmoid colon resulted in torsion.

SUMMARY

A case of recurrent sigmoid volvulus has been presented which was initiated by a chronic anal fissure and aggravated by aerophagia with a tracheostomy. Fissurectomy removed the trigger mechanism and has prevented any further abdominal distention, partial large bowel obstruction or volvulus.

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STUDIES ON THE EFFECT OF CORTISONE ON SPECIFIC GASTROINTESTINAL FUNCTIONS: THE SYSTEMIC RESPONSE TO INJURY

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Previous studies have demonstrated the totality of the body's response to major trauma.[3] The individual as an entity is injured and the individual, not just the wound site, responds as an entity.

Among the responses to injury, or perhaps reflecting responses to injury, numerous changes in the function of the liver,[6] gallbladder,[4] and pancreas[5] have been observed. Among the changes depicted by hepatic function tests, is a positive cephalin flocculation reaction. Within a few hours after an individual is injured, an increase in the flocculation of the cephalin-cholesterol emulsion can be demonstrated. The magnitude and duration of the abnormality apparently varies with the magnitude of the injury, but it is not unusual for the flocculation to be increased for 5 to 7 days.

Similarly, cholecystographic studies often result in nonvisualization of the gallbladder during the first week after injury. Thereafter, a normally functioning gallbladder may be demonstrated by repeated study.

Simultaneously, the battle casualty often demonstrated a sharp reduction in his plasma amylase activity for the first few days after injury.

The above phenomena were repeatedly observed in casualties with injuries of various parts of the body. Because the mechanism of these changes has not been identified, the following study was undertaken in an effort to isolate one of the mechanisms of the response to trauma.

METHODS

Six healthy, young, men served as volunteers. Control studies were obtained on the first and second day. On the third, fourth, and fifth days, each patient received by mouth, 200 mg. of cortisone daily. The observations were repeated on the sixth day after which the experiment was terminated.

Serum amylase activity was determined daily, utilizing the method of Wohlgemuth as described by Bray[1] in which the normal range in this laboratory (70 controls) has been *15* to *70 units* per 100 ml. of serum. Cephalin flocculation was determined daily by the method of Hanger[2] in which the normal reaction is graded zero to plus 2.

Supported in part by a grant from the Department of Army (DA-49-007-MD-732).

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Cholecystograms were performed by the standard oral technic utilizing Telepaque (3.0 grams). This study was performed twice, the control study was performed prior to the administration of cortisone. In the second study, the subject received the Telepaque on the final day of cortisone administration; his roentgenograms being taken the following morning.

RESULTS

The results are recorded in table I. In no instance was there an appreciable change in the cephalin flocculation reaction nor in the serum amylase activity. The gallbladder of one subject failed to visualize during the control period and so was not restudied. The other 5 subjects revealed a normal gallbladder during the control period and their cholecystographic visualization remained unchanged by cortisone.

Whatever the mechanism or mechanisms of the physiologic alterations depicted by the cephalin flocculation, serum amylase determinations, and by cholecystography in the injured man, the alterations are not reproduced by the

TABLE I

Subject	Study	Day of Study					
		1	2	3	4	5	6
				Period of cortisone administration			
1	Cephalin flocculation	1+	—	1+	1+	0	0
	Serum amylase	33	40	41	29	29	22
	Cholecystogram	—	Normal visualization	—	—	—	Normal visualization
2	Cephalin flocculation	0	1+	0	0	0	0
	Serum amylase	39	17	31	50	45	46
	Cholecystogram	—	Nonvisualization	—	—	—	—
3	Cephalin flocculation	2+	0	—	0	1+	1+
	Serum amylase	58	66	52	52	76	70
	Cholecystogram	—	Normal visualization	—	—	—	Normal visualization
4	Cephalin flocculation	2+	0	0	0	0	0
	Serum amylase	58	47	44	34	36	—
	Cholecystogram	—	Normal visualization	—	—	—	Normal visualization
5	Cephalin flocculation	2+	0	0	0	0	1+
	Serum amylase	—	50	47	66	77	75
	Cholecystogram	—	Normal visualization	—	—	—	Normal visualization
6	Cephalin flocculation	1+	0	0	1+	0	1+
	Serum amylase	34	22	27	35	31	41
	Cholecystogram	—	Normal visualization	—	—	—	Normal visualization

administration of 200 mg. of cortisone for 3 days. This would suggest that the specific changes under study did not result from a comparable corticoid response.

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TREATMENT OF ACUTE INTESTINAL OBSTRUCTIONS

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The treatment of acute intestinal obstruction has undergone several major changes in the past 50 years. During this period mortality figures have also changed from 60 per cent in 1908,[10] to an over-all mortality of 6.6 per cent[3] in 1956. Much of the credit for reducing the mortality to its present level must be given Wangensteen and his associates.[12] They entered upon a study of bowel obstructions when operative mortality approached 45 per cent, and succeeded in lowering this figure to 17.9 per cent. Intestinal intubation became an accepted therapeutic weapon and offered a means of avoiding surgery on an ill, depleted and toxic patient. In recent years the tendency has been toward early operative decompression of the obstructed intestine, and more and more surgeons concerned with the problem are recognizing early surgery as the treatment of choice in acute intestinal obstruction.

Reasons for the upsurge in operative treatment parallel the advances made in other surgical fields and include the advent of effective antibiotics, knowledge of abnormal electrolyte patterns, less fear of peritoneal contamination, improved anesthesia and blood transfusions, to mention only a few. Advances in surgical physiology have led to a better understanding of the factors and processes existent in the obstructed intestine, and a clearer perception of the proper treatment of obstructions.

It is a matter of importance that the treatment of acute intestinal obstruction be clarified and a definite course decided upon. Recent figures[3] indicate that the incidence of acute adhesive ileus has nearly doubled in the past 5 years. Most authors now report intra-abdominal adhesions as accounting for up to 90 per cent of all acute small bowel obstructions. External hernias, once leaders in the production of obstructions, now account for only 10 per cent or less of the total. In view of the increased volume of elective abdominal surgery being performed, it seems reasonable to assume that the future holds an even greater increase in the incidence of acute bowel obstructions.

With a few exceptions that will be detailed later, it is thought that all acute bowel obstructions should be operated upon, as soon as the diagnosis is certain and the patient adequately prepared. Intestinal intubation as a primary therapeutic device reflects an era when the surgical mortality in acute intestinal obstruction was prohibitive. It has little place in the treatment of obstruction today, except as a preoperative measure and in some selected postoperative patients. A short nasogastric tube is recommended during the preoperative period, not as a method of therapy but as an adjunct to operative intervention.

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This paper was entered in the Scientific Award Essay Contest, The Southeastern Surgical Congress 1957.

It is believed that the hazards of an unrecognized strangulated obstruction treated by suction are greater than the risks of operation. The mortality for intestinal obstruction as a whole has decreased markedly, but the mortality for strangulated obstructions is still approximately 30 per cent. This is a reasonable argument for operative relief of all obstructions and should help lay the ghost of nonoperative treatment to rest. Much emphasis has recently been placed on reliance upon the development or presence of the so-called *"signs of strangulation"* as an aid in differentiating which obstructions may be safely treated by suction and which should be operated upon. Becker[1] found that only an occasional patient with a strangulating obstruction failed to exhibit at least one of these signs (fever, tachycardia, tenderness, leukocytosis or palpable mass), but that the nonstrangulating obstructions also exhibited one or more of the same criteria. The decision to treat such a patient conservatively places a grave responsibility upon the clinician.

Figure 1, taken from Bollinger,[3] illustrates graphically the rise in mortality with delay in operation. Much of this delay is due to unavoidable factors. Delay in seeking medical advice, delay in diagnosis and delay in referral to a surgeon are regrettable but at present must be accepted. The use of long intestinal tubes as a primary method of treatment is considered an avoidable delay in operative therapy. Intestinal intubation, even in the hands of those expert in the method, is occasionally allowed to lessen the patient's chances of survival. Valuable time is consumed in attempts to pass the tip of the tube past the pylorus. Even passage of the tube past this critical sphincter is no guarantee of successful decompression. The surgical adage, *'when the bowel sounds are gone, you've waited too long'*, was never more true than in these patients. Absence of peristalsis will prevent further passage of the tube and attempts to force it distalward are useless. Abuse of tube decompression, as noted by Becker[2] is a major factor in the continuing high mortality of strangulated obstructions.

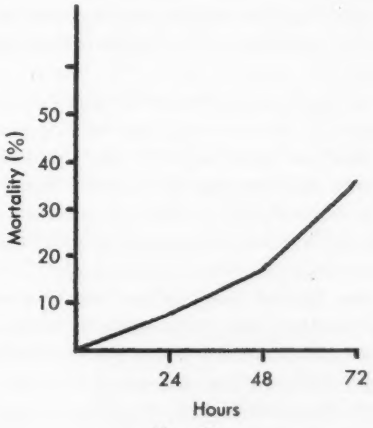

FIG. 1[3]

Exceptions to a hard and fast rule of operative decompression are existent. These include intussesceptions reduced by barium enema, and volvulus of the sigmoid reduced by proctoscopic examination or barium enema. Reduction of intussusceptions by barium enema should not be attempted by anyone not thoroughly familiar with the technic, and the surgeon must be present at the attempted reduction. Reduction of volvulus by proctoscopic examination is a procedure beautiful in it's simplicity, and when it occurs is the ideal solution to a dangerous situation.

Use of the long intestinal tube is justified in those acute obstructions occurring in the early postoperative period. Here, used in a patient already depleted by prior surgery, a trial of intestinal intubation is a worthwhile endeavor. The special circumstances surrounding this particular entity consist of fresh fibrinous adhesions, combining with a varying degree of ileus to form a picture of acute intestinal obstruction. This combination is most often met in the postoperative ruptured appendix; however it may occur after any abdominal procedure. A trial of suction, after fluoroscopic control of duodenal intubation is successful, is often followed by dramatic relief of distention, passage of flatus and cessation of vomiting. The patient must be carefully watched and abdominal films taken at 6 to 8 hour intervals. If relief is not marked within 24 hours, or if distention increases within 12 hours of duodenal intubation, surgery must be performed.

Preoperative Preparation: Under the usual conditions, 6 hours is deemed sufficient time to prepare the obstructed patient for surgery. If strangulation is suspected, or if a closed loop type of obstruction is present this interval is shortened accordingly. Upon admission erect and scout films of the abdomen and a chest plate are taken. A routine blood count and urinalysis are performed and electrolyte determinations are ordered. Other studies such as an electrocardiogram, proctoscopic examination or barium enema are performed as necessary. An anesthesia consultation is usually obtained, particularly in older debilitated patients and infants. Obstructed patients are often much more ill than appearances would indicate and one must use the most delicate judgment in their preoperative management.

Dehydration: Most obstructed patients are dehydrated to some extent. Patients with early cases will perhaps show no such derangement and in these patients too aggressive fluid replacement is apt to do more harm than good. Clinical dehydration presupposes a loss of at least 6 per cent of the body weight in fluids, or 4200 cc. in a 70 Kg. man. Up to 3 per cent of the body weight in fluid may be given in the first hour if necessary. The balance can be given during surgery or in the ensuing 4 to 6 hours if desired.

Shock: The obstructed patient may exhibit varying degrees of shock. Subclinical shock and hypovolemia are quite common, particularly in infants and the aged. This may be manifested only by a postural hypotension, but must be watched for and treated. Dehydration may mask an actual anemia. Whole blood must not be withheld if there is the slightest indication for its administration.

Electrolytes: Many obstructed patients exhibit some degree of electrolyte

imbalance. Patients with early cases will usually show normal values, but many of the later ones have been vomiting for days prior to admission and will show significant deviations from the normal. Obstructed bowel retains little effective resorptive power and even in the presence of minor quantities of vomitus may produce marked deficiencies. Correction of existing deficits should be begun upon admission. A prompt and accurate emergency laboratory will aid in intelligent replacement of losses and correction of imbalance. Surgery should not be withheld until normal values are obtained, however.

Distention: Preoperative distention is combatted by insertion of a nasogastric tube. No attempt is made to pass a long intestinal tube. Relief of gastric distention is all that is expected or desired preoperatively. No attempt is made to judge the progression of the distention by serial films unless the diagnosis is in doubt. In the usual case the patient has been operated upon, the obstruction relieved and the intestines deflated by the time the next set of films is due.

Infection: Infection plays a major role in the mortality of bowel obstructions. Patients are placed on penicillin and streptomycin in the short preoperative period, and continued on these drugs after surgery. Recent work by Cohn[4] would indicate that the course of strangulation obstruction in experimental animals can be favorably influenced by the administration of broad spectrum antibiotics, notably Aureomycin and more recently Achromycin, and therefore one of these is used intravenously in the preoperative and immediate postoperative period.

SURGICAL TREATMENT

Large Bowel Obstruction: The merits of early operation in large bowel obstructions are not disputed as soon as a reasonable diagnosis is made, either by X-ray, rectal or proctoscopic examination, or emergency barium enema, and the patient is adequately stabilized.

A transverse or sigmoid colostomy is employed for decompression of left-sided lesions. If the bowel is found to be unmanageably distended by gas, a needling procedure as described below is employed to relieve the distention and facilitate performance of the colostomy. The loop of colon, deflated and infinitely more manageable is then brought out through the wound and the colostomy established by whatever means the operator favors.

In the management of the occasional obstructing lesion of the right colon or hepatic flexure, an adequate cecostomy is employed, in which the cecum and ascending colon are mobilized sufficiently to allow exteriorization of the circumference of a portion of the wall of the cecum. Again if distention is a major factor, needle decompression is employed. Occasionally, in the presence of open perforation or gangrenous bowel, an exteriorization procedure is necessary. In favorable right-sided lesions in good-risk patients, a primary resection of the right colon is sometimes employed. Rarely a bypassing ileotransverse colostomy is utilized.

Small Bowel Obstruction: It is in small bowel obstructions that confusion in treatment exists, and in these cases early operative management is strongly advocated, as soon as the diagnosis is certain and the patient adequately pre-

pared. Uncontrolled distention is one of the most lethal factors in intestinal obstruction and decompression of the distended loops at operation is an essential part of the surgical procedure.

Aseptic decompression of distended bowel by a tube at operation was advocated by Monks as long ago as 1905.[7] Since that time many surgeons, notably Moynihan[8] and Wangensteen[11] have urged the use of aseptic enterotomies to relieve distention. Lord Moynihan has stated, "No operation for acute obstruction can be considered complete which leaves an intestine overdistended by contents of an offensive and poisonous nature." Gas and fluid, if allowed to remain within the bowel lumen, may have to pass through a recent anastomosis and may not be expelled for days after operation. Deflation at operation must be accomplished if the patient is to have a smooth postoperative course. Closure of the incision is facilitated, possible absorption of toxic products is avoided, better respiratory exchange is possible, and the risk of postoperative dehiscence is materially lessened by adherence to this rule.

Individual preference for the various methods of decompression at operation will govern the course followed. The simpler methods are more attractive. For preliminary deflation of intestine containing primarily gas, the use of tangentially inserted needles of no. 18 to no. 14 gauge attached directly to the operating room suction tube, or more tightly fitted with an adapter, is satisfactory.[5] Peritoneal soilage is avoided if the needles are carefully inserted bevel down in an area of relatively normal bowel and forced distalward in the subserosal-muscularis plane. A definite cleavage plane is encountered at this level, and a distance of 1 centimeter or more is traversed before the needle is allowed to enter the lumen. Upon completion of the aspiration, the needle is withdrawn and the tangential opening effectively sealed by pressure with thumb and forefinger. If more than one aspiration is performed, as is usually necessary, fresh needles are used. In large bowel the aspirations are best performed through one of the taeniae.

Table I gives the results of a recent investigation[5] of the effects of multiple needle aspirations on dogs subjected to both artificially induced distention and mechanical obstruction. A total of 276 punctures were performed on 25 animals with no evidence of resultant peritonitis. It is thought that multiple needle aspirations, properly performed, are innocuous and helpful in relieving distention at operation. Multiple needle aspirations are not entirely satisfactory for fluid, but serve as a useful adjunct in operative deflation, particularly in gas filled large bowel obstructions.

Most small bowel obstructions can be effectively aspirated by selecting an area of relatively normal bowel and inserting either a sterile Levin tube or the ordinary operating room suction tube through a small enterotomy opening. With careful packing, spillage should be minimal. By gentle stripping between the index and forefingers, and by careful manipulation of the distended loops, one can move the gas and fluid to the aspiration point and safely flatten the intestines. The hazards of bowel stripping have been enumerated by others,[9] but if done gently it is a definite and distinct aid in performing the aspirations. No ill

TABLE I[5]

Summary of results—aseptic needle decompression

Distention	No. Animals	Punctures	Mortality	Autopsy
Small bowel				
Artificial...	9	111	1 (26 days) distemper	No evid. peritonitis (3)
Obstruction .	6	30	1 (6 days) distemper	Local infl. ligature site (1)
Large bowel				
Artificial...	8	124	0	Adhesion of omentum to serosal tears (2)
Obstruction .	2	11	0	No evid. peritonitis (1)
Total.....	25	276	2	7

effects have been noted either in experimental animals or in patients. Upon completion of the decompression, the small enterotomy is closed, either by means of a previously placed purse string suture or interrupted intestinal sutures.

In some patients with markedly distended loops, release of the obstruction is facilitated by first decompressing the intestines and then seeking the obstructed point. This is particularly true in the markedly distended patient where sudden evisceration of the coils of dilated tense bowel is apt to produce significant degrees of shock. By first decompressing the bowel, one is able to increase the available working space and minimize handling of the bowel. Resections, if necessary, are easier to perform on deflated intestine. If bowel of questionable viability is present it is important to first decompress before attempting an attack on the obstructing mechanism, as there is a possibility of spillage produced by the weight of the filled loops perforating the precarious bowel. Where doubt exists as to viability, hot moist packs are applied and bowel not showing signs of returning circulation after 15 to 30 minutes is resected. Block resection of contaminated fields, particularly gangrenous femoral hernias and difficult intussusceptions is advocated.

Postoperative Care: General principles of postoperative care of any abdominal operation apply as well to intestinal obstructions. One should expect the period of ileus to be somewhat prolonged. Meticulous attention to the proper functioning of the nasogastric tube is mandatory if postoperative distention is to be avoided. Many of these patients under conditions of fear or stress become temporary aerophagics and swallow tremendous quantities of air. If the nasogastric tube is functioning properly, this is of no concern, but even failure of the tube for a 6 hour period in such a patient will result in substantial distention. Cautious liquid feedings are instituted upon return of healthy bowel sounds. Fluids and electrolytes are maintained intravenously in a conventional fashion.[6] Antibiotics are used routinely on these patients. Penicillin and streptomycin are used, and if peritoneal soilage has been significant, intravenous broad-spectrum drugs such as Achromycin are continued for 24 to 48 hours after operation.

SUMMARY AND CONCLUSIONS

The treatment of choice for acute bowel obstruction is early surgery, as soon as the diagnosis is established and the patient adequately prepared.

Decompression of distended bowel at operation is an important and integral part of the operation.

Multiple tangential needle aspiration is a simple and safe means of reducing gaseous distention, and enterotomy the method of choice in relieving combined gaseous and liquid distention.

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EDITORIAL

HOMOTRANSPLANTATION

The surgeon, ever looking for new worlds to conquer, dreams of the day when it will be possible to transplant living tissue from one individual to another. Currently many tissues are transplanted in the human being, but with the possible exception of the cornea, the transplant does not survive. The technical problems concerned with transplantation of whole organs were solved by Alexis Carrel 50 years ago, when he successfully transplanted the kidney. Since that time virtually every organ has been transplanted from one animal to another, and function will occur for a period varying from a few days to 2 or 3 weeks, following which time function ceases and the organ dies. Although an occasional dissenting opinion is expressed, most investigators agree that rejection of homotransplanted tissue represents an immunologic process—although it is difficult to demonstrate antibodies against the donor tissue by the usual immunologic technics and there are no consistent changes in the gammaglobulin fractions of the blood. The immunologic concept of tissue rejection is supported by the fact that a second organ transplant is rejected more rapidly than the first, the observation that successful homotransplantation may be accomplished in patients with agammaglobulinemia, and by reports of successful transplantation in patients who are identical twins.

Attempts to alter the immunologic response by irradiation, the use of drugs, and administration of hormones have provided a great deal of interesting information, and an occasional report indicates that the use of these agents may prolong the life of homotransplanted tissue. However, there have been no permanent survivals of adult organ transplants, and none of the agents now available have proved successful enough, within the limits of safe dosage, to warrant investigation in the human being.

Transplantation of fetal tissue has occasionally been successful both in the experimental animal and in the human being. Animal experimentation indicates that the ability to accept foreign tissue is lost with age, and suggests that tissue specificity is acquired after birth. Transplantation in the human being usually has consisted of implanting small bits of endocrine glands, notably adrenal and parathyroid, into muscle. There are only three reported cases of full organ transplants of thyroid and parathyroid. Long term survival resulted in two. Further investigation of transplantation of fetal endocrine glands seems indicated, for small glands can supply the full requirements of the adult. Unfortunately, most of the other organs are too small to be of real benefit to the adult; furthermore, the supply of suitable fetal tissue is small, so that this approach to the problem has limited clinical application.

The most promising field of investigation concerns attempts to produce "*immunoparalysis*". There is evidence to indicate that the injection of antigens derived from prospective donor tissue may produce a state in which the recipient

will fail to respond to further injections of this antigen. At such a time, successful homotransplantation may be accomplished in the experimental animal. This concept is supported by the observation that the injection of tissue from a prospective donor into a fetus will allow successful homotransplantation after birth of the treated animal. It appears that the factor which is responsible for rejection of transplanted tissue is common to all tissues of the body; consequently, it may be possible to produce an antigen of tissue which is easily available, other than that to be used for transplantation.

These problems are being intensively investigated in many centers, and it seems likely that in the future patients will have the opportunity to trade old organs for new.

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BOOK REVIEW

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted with solicitation.*

The Clinical Management of Varicose Veins. By DAVID W. BARROW, M. D. Hoeber-Harper Second Edition; 169 pages.

This monograph offers precise and complete coverage in regard to the clinical management of the age old malady of varicose veins. The text is based primarily upon the author's clinical experience, but other methods and concepts are discussed and evaluated. It is quite readable and is profusely illustrated with drawings, diagrams, photographs, and case reports. The author is quite thorough in describing the various operative and nonoperative procedures with specific directions as to how each is performed. There are twelve chapters dealing primarily with anatomy, physiology, pathology, symptomatology, examination of the patient, treatment by injection, surgical treatment (to include long and short saphenous systems and incompetent communicators), complications, thrombophlebitis, and aftercare. An excellent bibliography is given for each chapter. Two points stressed in the proper treatment are: (1) the necessity of complete obliteration of all varices, and (2) the ad infinatum follow-up on all these patients. While this book fails to offer a new panacea for varicose veins, it does offer an excellent reference for the present day concepts of this problem.

ROGER D. SCOTT, M. D.

TRACHEOTOMY: A CLINICAL AND EXPERIMENTAL STUDY

PART I*

THOMAS G. NELSON, MAJOR, M.C., U. S. ARMY

CHAPTER I. INTRODUCTION

Tracheotomy is an operation of unusual historic interest. From the time of the earliest fragmentary records of tracheotomy, before the birth of Christ until the early part of the nineteenth century, the procedure was considered hazardous and was rarely performed. Although certain improvements of technic and instruments came about after 1825, the indications for and timing of the operation remained hazy until about 1900. Tracheotomy frequently was reserved for drastic emergencies—delayed until the patient was moribund and then administered, as is sometimes the case with oxygen, as a last rite to the dying. The early death of the patient after tracheotomy often was attributed to the operation itself, rather than to the disease for which it was performed or the delay in its performance. This attitude did much to crystallize the belief that tracheotomy was difficult to perform, attended by many pitfalls, and frequently yielded poor results. Only during the past two decades has the operation assumed its rightful place as a safe, simple procedure with a wide range of usefulness. The historic aspects of these changes in the uses of tracheotomy is a fascinating study in itself. A compendium of the historic events in the development of this invaluable surgical procedure follows in Chapter II.

In ancient times the operation was used for a few types of inflammatory obstructions about the pharynx, neck, and floor of the mouth. After the Renaissance, the indications were expanded to include trauma and foreign bodies of the airway and diseases of the larynx, such as diphtheria, syphilis, and tuberculosis. Except for recent years, much of the modern use of the operation has been guided by the writings of Chevalier Jackson. Laryngeal obstruction became recognized as the chief indication for tracheotomy and special care was taken to avoid performing the operation unless mechanical obstruction of the airway was present. Medical thinking became stereotyped, tracheotomy became synonymous with laryngeal obstruction, and few constructive ideas concerning new uses for the operation were advanced.

In recent years there has been a considerable shift in emphasis regarding the indications for tracheotomy with recognition of the more physiologic and functional indications in addition to those of a strictly obstructive nature. Largely this has come about through demonstration of the value of tracheotomy in bulbar poliomyelitis, head and chest injuries, and other conditions wherein

* *Editor's note:* This report is an invaluable contribution to the field of tracheotomy. In recognition of that value, it is being presented to our readers with the anticipation that in addition to being an interesting review of the subject, it will clarify problems of technic. Because of the length of the manuscript, it will be presented in three consecutive issues of The American Surgeon—July, August, and September.

normal respiratory efficiency is impaired because of the patient's inability to maintain normal ventilation and control of secretions.

In order to determine if these changing concepts are reflected in the present-day use of the operation, a clinical analysis was made of 300 consecutive, unselected cases in which tracheotomy was employed. This study, to be presented in Chapter III, serves to reflect the recent trend of new ideas and uses for the operation. In addition, during the course of this study, certain indications and practical uses for tracheotomy were uncovered which are not believed to be widely known.

Through past centuries there has been considerable variation in the technic for making an incision into the trachea. In ancient days the trachea was entered between the cartilaginous rings because of the fear that divided cartilage would not heal. In the eighteenth century vertical incision through several tracheal rings became the custom once it was learned that the trachea would heal if the cartilages were severed. In the nineteenth century the removal of a piece of the anterior tracheal wall was first advocated as a means of dispensing with the tracheotomy tube, but later came to be utilized in cases where the cannula was employed. The cruciate and T-shaped incisions into the trachea are of more recent origin, and as yet it has not been determined when they first came into use. Analysis of the technic in the 300 cases just mentioned revealed that all these methods were employed. Although the clinical study revealed many advantages of the excision technic, the vertical incision proved to be the most popular choice.

Certain aspects of these technical variations of tracheotomy and their relationship to complications and eventual healing have been studied previously in a limited fashion, but in no instance heretofore has a clinical or experimental comparison been made of these methods of incising the trachea. These factors provided the stimulus for one of the major objectives of this study: to determine, through clinical and experimental investigation, the relative merits of the various technics of opening the trachea.

Early in the history of this operation, the fear of severe hemorrhage when the thyroid isthmus was incised led to the unfortunate concept of high and low tracheotomy. Unhappily, it was quickly learned that the operation above the thyroid isthmus, either through or above the cricoid cartilage, was faster, easier and less bloody. The high operation became known as cricothyroidotomy, or superior tracheotomy. During the latter years of the nineteenth century there was much controversy over the advantages and disadvantages of the two types. Although it was soon discovered that operations through or near the cricoid cartilage yielded a high incidence of cicatricial stenosis, cricothyroidotomy continued in wide usage. After the report of Jackson[142] on the dangers of the high operation this technic became widely condemned. In recent years there has been renewed interest in cricothyroidotomy as a substitute for emergency tracheotomy in certain emergencies. It has also been suggested recently that cricothyroidotomy, because of its simple technic, might be used in combat or mass casualties.[293] Thus, it seems that, despite the deservedly infamous reputation of cricothy-

roidotomy in the past, there is continued clinical use of the operation and there are hints that it may become more commonly used in the future. These facts largely supplied the stimulus for the last major objective of this study; namely, to determine by experimental investigation the relative merits of the various locations that can be used for an artificial opening of the airway.

Briefly summarized, the major purposes of this study are:

1. To present the story of the evolution of tracheotomy, leading to its presently accepted position as a valuable procedure with a wide range of applicability in many fields of medicine.

2. To present an analysis of the indications, technic and complications in 300 patients in whom 310 tracheotomies were performed, and to emphasize certain lesser known clinical uses of the operation.

3. To present a clinical and experimental evaluation of the technical aspects of the operation with the view in mind of determining the optimum method and location for an artificial opening into the trachea.

CHAPTER II. HISTORIC BACKGROUND

Tracheotomy before the Time of Bretonneau (1825)

Although detailed historical investigations have been done,[55, 56, 113, 122] as yet it cannot be determined exactly when and by whom tracheotomy was first performed. It is known that many ancient and early writers who described the operation, even in detail with diagrams, did not actually perform it. Medical writings prior to the Renaissance consisted many times of fragmentary sketches, excerpts, and paraphrases beset with language and translation difficulties. To Dr. F. J. Collet[55, 56] and to Dr. E. W. Goodall[113] must go much of the credit for unraveling this aspect of the story of tracheotomy. Much of the material presented here is taken from Dr. Goodall's clear and exhaustive study.[113]

The word "trachea" is derived from the Greek language and originally meant "rough."[113] The trachea itself was known as the tracheia arteria, or "rough artery." Other designations for the trachea included: the arteria aspera or "artery of air," "the bronchus," "the artery," or "the larynx." After the Christian era, tracheia arteria continued to be the most popular designation for the windpipe. Later, the word "arteria" was dropped and the structure became known as the tracheia. In ancient writings, "laryngotomy," pharyngotomy," and "perforation of the tracheia arteria or arteria aspera" all referred to the operation of opening the windpipe.[113]

Later (800–1200 A. D.), from Latin translations of certain Arabian medical writers, the trachea was known also as the canalis pulmonis, or "canal to the lungs." The operation was called incisio cannae pulmonis, or "opening of the windpipe." Occasionally the windpipe was referred to as "the guttur." After the Renaissance the operation was called laryngotomy, bronchotomy, and finally tracheotomy.[112]

The only indication for tracheotomy in ancient and medieval times was cynanche or synanche which referred to a group of miscellaneous inflammatory processes about the floor of the mouth, larynx, and neck.[113] Cynanche is believed

to be derived from the Greek word, *kunangche* (*kunes*, dog), meaning "affliction of the dogs," because of the analogy between the respirations of those affected and a panting dog. Cynanche was restricted by Galen to laryngeal and pharyngeal inflammations, while synanche was related to faucial infections. A variety of what are now considered distinct disease entities were included in these terms.[55, 56]

Tracheotomy is first discussed by Galen (131–201 A. D.) and Aretaeus (117–138 A. D.), neither of whom professed to have performed the operation; indeed, Aretaeus was much against it, believing that the cartilages would not unite. Both of these writers credit the operation to Asclepiades of Prusa, who lived about 124 B. C. A few fragments of the writings of Asclepiades have survived, but in none is there any mention of tracheotomy.[65, 113] According to Galen, Asclepiades operated in cases of cynanche or synanche. Aretaeus described two forms of this disease, one of great inflammatory swelling of the pharynx and the back of the mouth (perhaps retropharyngeal abscess), the other a collapse of these parts with inward compression and greater suffocation. The latter form, says Aretaeus, is the one in which some surgeons opened "the artery." The operation was reserved until the patient had reached extremes of suffocation.[113]

The second person said to have performed the operation is Antyllus (second century A. D.). Paul of Aegina (625–690 A. D.) quotes from a work of Antyllus, suggesting that Antyllus not only described the operation, but actually performed it. Antyllus, who called the operation "pharyngotomy," advised that the "arteria aspera" should be opened at the third or fourth ring—clearly tracheotomy as we know it today.[55, 113] The patient was positioned erect with the head back, to bring the windpipe into better view, and a transverse skin incision was made to conform with the incision into the trachea. Still quoting Antyllus, Paul notes that the windpipe is known to be entered by the rush of air and loss of voice. From this description is is apparent that the operation was performed with the patient sitting. This was the only position used until the time of Moreau (seventeenth century), who was the first to place the patient in the recumbent position.[113]

Caelius Aurelianus (fifth century A. D.) described the operation as the "futile and rash invention" of Asclepiades and indicated that by his time it was regarded as "fabulosa," taken to mean that it was more talked of then done. Aurelianus called the operation "division of the artery, or laryngotomy."

Little more is heard of the operation until Rhazes of Baghdad (860–932 A. D.) writes that he saw a medical man named Ancilisius open the "canalis pulmonis," or windpipe. Further details of the operation are not known except that the windpipe was incised between two rings.

Albucasius of Cordova, Spain (1013–1106), who wrote a medical text based largely on the works of Paul of Aegina, demonstrated, by sewing up the trachea in a servant-maid who tried to commit suicide, that the trachea would heal when incised. This case greatly impressed subsequent writers for it is often quoted.[221]

Avenzoar (1126–1198) wrote that, while a student, he was anxious to refute the beliefs of certain persons of his time that Galen had stated that the operation could not be done. To prove that tracheotomy was possible, he successfully

performed the procedure on a goat. Avenzoar was one of the few men in medieval medicine who dared to dispute Galenism.[107]

Benivieni (1440–1502) is stated to have attempted tracheotomy on a patient with "angina," but whether the operation was completed is not known.[188]

There are scattered writings indicating that Homer wrote of tracheotomy and that Alexander the Great performed the operation on one of his soldiers with the point of his sword,[114] but these instances are based more on fable and inference than on verifiable information. Even if we allow that any or all of these ancient operations took place, little is known of the reason for the operation and nothing is known of a successful outcome.

The first clear account of a successful tracheotomy was recorded in 1546 by Antonius Musa Brasavola (1500–1570). He told of opening the windpipe and saving the life of a patient near death from angina and an abscess which was obstructing the "canalis pulmonis" or trachea. Brasavola was justifiably proud of this case, particularly since the barber-surgeon in attendance was afraid to do the operation. The patient was visited by the Duke of Ferrara so that he might see the tracheal opening. Brasavola also remarked on the case of a cowherd who recovered after attempting suicide by cutting five rings of his trachea and part of the larynx. This case also demonstrated that divided tracheal cartilages would unite.[55, 113, 221]

After Brasavola's case was recorded, Fabricius of Aquapendente (1537–1619) wrote at some length on the technical aspects of the operation.[113] It was known in his time as the "scandal of surgery" and was regarded by the profession with fear and trembling. Although he conceived that the operation should not be too difficult for the skilled anatomist, he declined to perform the operation, partly because of lack of experience and knowledge of anatomy and partly because of fear of loss in reputation should the patient die. The writings of Fabricius and his student Casserius (1545–1605) were the first clear descriptions of the operation after the time of Antyllus.

Although Fabricius did not perform the operation, he initiated several changes in terminology and technic. He was the first to drop the word "arteria" and refer to the structure as the tracheia alone, and he was also the first to advise a vertical skin incision and use of a cannula. Fabricius condemned the transverse skin incision because of danger of cutting the neck vessels and because it did not allow the surgeon to see enough of the trachea. He advised that a vertical line be marked with ink in the midline of the neck before the incision was made so that injury to the blood vessels could be avoided. The median vertical skin incision became the custom after this. Fabricius described and made drawings of the first tracheotomy cannula. The tube recommended by him was small, straight, and short, with two wings at its outer end to prevent it from slipping into the trachea. Tapes were tied to the tube under the wings and around the neck. The short length was to prevent injury to the posterior wall of the trachea, and the small size was to prevent too much air from entering the lungs. He advised that the cannula be placed between the cartilaginous rings and allowed to remain in place three or four days. With his newer terminology, vertical incision, and use

of a cannula, Fabricius was startlingly close to certain modern concepts of the operation.

Casserius, who called the operation "laryngotomy" but also did not perform it, likewise devised a tracheotomy cannula. His tube was of curved shape, made of silver, and perforated in the lower portion to allow better passage of air. Although Casserius did not draw attention to the shape of his cannula, it was of better anatomic curvature than those to follow, which were curved in the shape of a quarter circle. These quarter-circle tubes made of gold, silver, or lead were commonly used in the nineteenth century until subsequent revisions came about.[113]

Nicholas Habicot (1550–1624) in 1620 described the operation under the name of bronchotomy and was the first writer after Brasavola to report a successful operation.[56] He described four successful cases in a book of 108 pages confined to an account of the operation, the first of its sort.[56, 113] Habicot also devised a cannula of curved shape, and flattened from front to back to conform with the space between the cartilages.[113]

After Habicot's report, 23 successful operations were recorded over the next 200 years prior to Bretonneau's performance of tracheotomy for diphtheria in 1825. Thus, there were only 28 verifiable successful cases up to 1825, according to Goodall.[113] As he states, some reports undoubtedly were overlooked, and certain unsuccessful cases, though recorded, were not included in his tabulation; but even twice this number of recoveries over a period of nearly 2,000 years is not encouraging.

The indications in the first 28 reported successful tracheotomies included synanche and other inflammations of the pharynx and larynx, such as laryngitis, quinsy, and croup.[113] In addition, a few tracheotomies were performed for airway obstruction from trauma and foreign bodies. Brasavola's case is considered to be typical of the indication of synanche. Three of Habicot's four tracheotomies were performed because of obstruction from trauma. In one of these, removal of a blood clot from the trachea is probably the first instance of tracheotomy for removal of a foreign body. Habicot's fourth case was that of a 14-year-old boy who swallowed a bag of gold coins for concealment. The bolus became impacted in the esophagus and obstruction of the trachea resulted. After Habicot performed a tracheotomy, he was able to push the packet of coins distally into the stomach and it was subsequently recovered. Chevalier's case of croup, in which a membrane was vomited by the patient, is considered by Goodall[113] to be highly suggestive of laryngeal diphtheria, although Bretonneau is credited with the first successful use of tracheotomy in the management of this disease.[56, 113]

During the period of 200 years prior to 1825 some rather unique and unusual uses for tracheotomy were suggested. Tracheotomy was recommended by Detharding (1714) and Heister (1739) for persons apparently drowned. In 1783, de Pouteau stated that by the use of tracheotomy the water could be removed and air could be blown into the lungs.[113] These statements are probably the first inklings that tracheotomy could be used for artificial respiration. A singular occasion for tracheotomy was that of Chovell, a young English surgeon, who persuaded a

condemned man to have the operation performed before he was to be hanged.[122] The patient was still alive when released after the prescribed time of hanging, but died moments later. Chovell apparently gained much fame among the highwaymen of London for this attempt to thwart the gallows.

Despite the early demonstrations of Brasavola and Albucasius that divided cartilage would heal, the transverse incision between the tracheal rings was used exclusively until Heister in 1718 recommended a vertical incision through several tracheal cartilages.[113] He reserved this technic, however, for the removal of foreign bodies and continued to advise the transverse opening for cases of angina. It was 1815 before anyone was so bold as to remove a segment of the anterior tracheal wall, as suggested by Lawrence for those cases in which the cannula kept slipping out of the trachea due to cough.[55, 113] Later, the suggestion by Diffenbach that a piece of trachea be removed to allow room for the cannula was regarded as heresy.[227] Diffenbach wisely argued that the portion of trachea adjacent to the cannula would become infected and heal by scar, so initial excision was justified.

Of equal popularity at that time were the straight and quarter-circle cannulas, usually made of silver. Some surgeons deliberately dispensed with the tube while others used makeshift tubes, such as quills or reeds, when the standard instrument was not available.[113] In 1730, George Martine, the first British tracheotomist,[122] first suggested that the "pipe" be made double so that the innermost might be safely and easily taken out and cleansed "without any molestation to the patient." He admits in his letters that the proposal was not his, but that of one of his ministers (nurse?). Martine also commented on the needless pain which some surgeons took in bandaging and stitching the wound, for he found it to heal easily without such special care within a few days after the cannula was out.[113, 122]

Before the time of Bretonneau in 1825, the operation continued to have mixed and confusing terminology. Gradually the word trachea became better accepted and the operation came to be called tracheotomy. As Goodall[113] states, Thomas Feyens (1567–1631) of Louvaine was probably the first to refer to the operation as tracheotomy. In 1718, Lorenz introduced the word tracheotomy into his surgical text, which was later translated into English. He recommended that the other terms should be discarded. John Quincy, in 1726, first inserted the word tracheotomy into a medical dictionary as a synonym for bronchotomy. No distinction was made between high and low tracheotomy, as far as terminology was concerned, until Desault (1744–1795) recommended that laryngotomy, or cutting above the cricoid cartilage, should be distinguished from tracheotomy, or cutting the trachea below this cartilage. Bretonneau (1778–1862) and his pupil, Trousseau (1801–1867), both called the operation tracheotomy and it has been known since by that name.[113]

After his successful tracheotomy in a case of diphtheria in 1825, Bretonneau published a paper on this disease, which he named and established as a distinct clinical entity. He showed that diphtheria was identical with certain forms of croup and malignant angina, and separate from other forms of sore throat, scarlatinum, quinsy, and angina—matters that had remained obscure since the

days of Aretaeus.[107, 113] Even after other physicians in Paris recognized diphtheria, tracheotomy was not looked upon with favor for this disease until Bretonneau and Trousseau had used the operation successfully many times.[107] In 1833 Trousseau reported that he had performed the operation some 200 times and had saved 50 children with advanced disease.[55] This report swung the tide of battle in favor of tracheotomy and a veritable outpouring of reports on new ideas, indications, technic, and instruments began—an avalanche that was not to slow for nearly 100 years.

Evolution of Tracheotomy since 1825

Bretonneau and Trousseau used both the straight and quarter-circle tubes, and each later changed to the double-lumen type.[113] The tube was passed into the trachea alongside the knife and later over a probe or stylet, the forerunner of the modern pilot. A pilot was first used by Bourdillat in 1852 in conjunction with a bivalve tube of his design.[55] Other bivalve tubes were constructed so that the two blades could be squeezed together for easier introduction of the cannula.[113] Trousseau originated the use of a dilator to spread the tracheal opening while inserting the cannula. His two- and three-bladed instruments are still in common use today.

Many new ideas concerning the cannula and technic were advanced. Cock[51] revived the trocar method of entering the trachea which had been used successfully over 100 years before by Bauchot in 1749.[113] Chassaignac (1855) invented a device with a pointed hook to fix the cricoid cartilage. The knife was guided by a groove on the convex aspect of the hook as it was passed into the trachea.[113] A variety of complicated tracheotomy cannulas with movable shields, adjustable lengths, and flexible shafts were devised. Most of these were found to be generally impractical and quickly became discarded. However, Rethi[225] still advocates a lengthy flexible tube devised by Konig for use in bypassing obstructions below the level of the opening into the trachea.

Parker, in 1880, recognized the unphysiologic shape of the quarter-circle tube and devised one of obtuse angle that more nearly conformed to the relation of the skin to trachea—a tube quite similar to the modern type and curved at almost the same angle as the one devised by Casserius over 175 years before. This tube was of such practical function that it continues to be used in England on children, according to Goodall.[113]

The cannula was further modified by Jackson after the beginning of the twentieth century.[152] Now there is simplification of the tube, with unnecessary frills eliminated, a standard shape with only one or two varieties of lengths and curvatures, and a selection of sizes to fit the needs of the individual patient.

Trousseau and Bretonneau used the conventional tracheotomy approach with vertical incision of the skin and trachea. Trousseau emphasized that the operation was less dangerous if performed early and with care and deliberation, but also cited the skill and judgment sometimes necessary to save lives with a timely emergency procedure.[113] As surgical skill advanced during this period, many innovations in technic were tried. Special methods of suturing the soft parts

around the tracheotomy opening were advocated. These technics were designed to hold the tissues apart and eliminate the cannula.[182, 215] Hall,[125] in 1848, invented a punch forceps for the removal of a small segment of trachea so that a cannula would not be necessary. In 1864, Porter[214] of Dublin reemphasized Diffenbach's ideas on the importance of making space for the cannula by excising a piece of the trachea.

The vertical slit continues to be the most widely used method for opening the trachea. Cruciate or T-shaped incisions are used mainly as a matter of personal preference. Waldapfel[278, 279] strongly recommends that a portion of anterior wall be excised, for certain well-conceived anatomic reasons, and has devised an instrument for this purpose.

As can be seen there remains some diversity of opinion regarding specific points in technic. Before the twentieth century high or superior tracheotomy was widely used because of its speed and simplicity as opposed to low tracheotomy, considered to be difficult and bloody.[254] The high approach, designed to open the trachea above the thyroid isthmus, was advocated in adults, whereas the lower incision was used more in children, because of a supposed difference in the anatomic location of the isthmus at different ages. High tracheotomy also included the operation now known as cricothyroidotomy, which was favored because of the ease of performance.[48] The controversy over which approach was best continued well into the twentieth century, until the late effects of high operation became sufficiently well known.[142]

The first real efforts at standardization of the indications, instruments, and technic were made by Jackson[139] during the early years after 1900. The technic advocated by Jackson[152] consisted of a vertical skin incision of generous size, strict adherence to the median line of the neck, and a vertical incision of the tracheal rings well below the cricoid cartilage. None of these phases were new in themselves but the consolidation of these steps into a single procedure, useful for both emergency and elective tracheotomy, and Jackson's insistence that this was the only safe method of performing the operation, were original. This technic is probably still the most widely used today because of its speed, simplicity, and safety. Jackson's major arguments were that there should be one type of tracheotomy adaptable for elective or emergency use. He advocated that such measures as a transverse skin incision, special care to avoid the thyroid isthmus, special incisions into the trachea, and, above all, incisions through or near the cricoid cartilage, should all be abandoned completely. Jackson's report[142] in 1921 on high tracheotomy as one of the chief causes of laryngeal stenosis was largely responsible for the acceptance of his views.

Despite the insistence by Jackson that none of these principles should be violated, many variations continued in common use. Cricothyroidotomy continues to be advocated by Waldapfel[279] and others[72, 132] as a means of ready access to the airway in special emergencies and is advocated by Rethi[225] for elective operation under certain circumstances. The transverse skin incision has been revived in recent years and is recommended for elective[23, 59, 94, 225] and even emergency operations,[23] because of the better cosmetic result.

The use of cautery to control bleeding during the operation was first advocated by Krishaber[160] in 1867. The wave of popularity for this technic quickly abated because of wound complications, and this practice was largely abandoned by 1880.

One of the reasons for the intensive search for a quick and simple technic of tracheotomy was the lack of anesthesia for these cases. Ether and chloroform, once discovered, became widely used as anesthesia for the operation, especially in children.[17, 33, 283] Many disasters were reported from the use of general anesthesia, chiefly because laryngeal obstruction frequently became complete as the patient went to sleep. Following the use of cocaine as a local anesthetic about 1884, Jackson[139] quickly became a staunch advocate of its use. After his writings on the dangers of general anesthesia, it was not generally used except in special instances.

Trousseau[268] was cognizant of postoperative complications following tracheotomy and wrote an article in 1858 devoted to the aftercare of these patients in which he emphasized the need for maintaining a clear airway. In 1852, Chassaignac[49] stressed the value of clearing the trachea of blood, mucus, and even the false membrane in diphtheria cases. He devised several types of instruments and devices for aspiration, as did Aubert[7] in 1869. Montgomery[191] in 1885 advised aspiration of blood, mucus, and pus with a catheter before the tracheotomy cannula was put in place. In the modern era of the operation, Jackson[153] and others[110, 200] have stressed the importance of a clear airway following tracheotomy and have recognized that the tracheotomy is not the objective in treatment, but only a means to the end of securing proper ventilation.

Complications during and after tracheotomy have been observed frequently during the past 125 years in the history of this operation. As early as 1848 Heyfelder[130] reported the interesting complication of death from air embolism, and by 1850 Sestiers[242] had compiled a list of accidents that might occur during the operation. Hemorrhage from the neck vessels and the thyroid gland was considered a great hazard during tracheotomy and fatalities were reported from this cause.[180] Fatal hemorrhage from rupture of an unrecognized aneurysm was reported by Pooley[213] in 1874. Hemorrhage following surgery was reported many times. The most common causes were: insecure ligature,[245] ulceration of the trachea due to an improperly fitting cannula,[15, 240] and erosion of the tube into the innominate artery.[254]

In 1874 Bouchet[28] described a case in which suffocation resulted from "vegetations" in the trachea following removal of the cannula. The findings in this case are remarkably similar to those reported in 1954 by Conley[60] in cases with encrustations in the trachea following tracheotomy.

Wound infection following tracheotomy was described many times. The usual infection was either an erysipeloid involvement of the skin or an abscess about the tracheotomy site.[27] Occasionally these abscesses were massive and extended into the mediastinum.[27, 96] Mediastinitis was greatly feared following tracheotomy because of the high mortality.[96, 254] In 1864, Sottas[249] is believed to have first reported a case of injury to the esophagus during the operation. This was

likewise a complication to be greatly feared because of the possibility of mediastinitis.

The interesting complication of cervical and mediastinal emphysema was first described by Wilks in 1860.[288] In 1882 Champneys[47] first reported mediastinal emphysema and pneumothorax following the operation. In 1898 Ellett[83] reported a case of massive subcutaneous emphysema of the entire body following the procedure. The mechanism causing pneumothorax was elaborated upon by Iglauer in 1915.[136] Further clinical and experimental work has been done by Goldberg[112] and Neffson[201] in 1942, by Forbes[92, 93] in 1943 and 1947, and by Bowden[30] in 1950. These workers have verified Champneys' original contention that mediastinal emphysema and pneumothorax resulted, not from pleural injury, but from the passage of air by way of the cervical fascial planes into the mediastinum. Because of wound infection and emphysema it gradually became the practice to leave the wound open, although Forbes[93] has recently shown that emphysema has little relation to suturing of the wound.

As early as 1825, the complications of difficult decannulation and laryngeal stenosis following the operation were noted. White[287] at this time recorded a case in which the cannula had been in place for over two years because of stenosis after removal of the tube. Delayed decannulation and laryngeal and tracheal stenosis were of frequent occurrence following tracheotomy due to injury to the cricoid cartilage, infection, improperly fitting tubes, and prolonged wearing of the cannula.[15, 54, 73] In 1873, Rydygier of Warsaw first described a case in which low tracheotomy had to be performed because of stenosis following laryngotomy. He was among the first to recognize the dangers of the high operation. Low tracheotomy has since become the first step that is employed in the treatment of chronic laryngeal stenosis.[85, 137, 263] However, the high operation, with prolonged wearing of the cannula, continued to be advocated and used[48] until well after 1921, when Jackson[142] made his classic report on laryngeal stenosis following high tracheotomy. Since Jackson's work the incidence of this complication has steadily decreased until, at the present time, causes other than tracheotomy are responsible for most instances of this condition. It is now rare to see this complication after tracheotomy.

During the period from 1840 to 1884 tracheotomy was recommended by Trousseau[268] and others[118, 248, 267, 281] for the treatment of chronic laryngeal diseases such as tuberculosis and syphilis. In 1841 Raphael[224] reported the use of tracheotomy in a patient with a submaxillary tumor, and in 1858 Birkett[20] recommended tracheotomy for all types of cervical tumors. The operation was found to be of benefit for bypassing tracheal compression from thyroid growths,[16] and numerous technics for the removal of foreign bodies were devised.[186]

During the Civil War (1861–1865), bronchotomy, which included 14 tracheotomies and 6 laryngotomies, was performed only 20 times to relieve airway obstruction.[187] This is an exceedingly low incidence for the operation, in view of over 12,000 head injuries, 10,000 maxillofacial injuries, and 5,000 neck injuries tabulated in the same report.[187] Of these 20 operations, only 6 were performed because of wounds, while 14 were done for diseases of the respiratory tract. Both low tracheotomy and cricothyroidotomy were described, and in one case the

technic of Chassaignac,[49] using the hook and groove instrument, was used. The mortality rate in the 20 cases was 60 per cent. Some patients died immediately after the operation from asphyxia or apnea, while others expired later of pneumonia.

Certain unique indications and uses of special interest were noted during the period from 1825. Tracheotomy was reported after sulfuric acid burn of the larynx[233] and for scalding of the larynx and respiratory mucosa.[115] In recent years there has been renewed interest in tracheotomy for this type of injury. Tracheotomy again was recommended for drowning cases,[185, 230] and cases were reported in which recovery occurred because of the prompt use of tracheotomy and artificial respiration after the patient was thought to be dead from asphyxia.[127, 155] In 1886 Bourneville[29] reported a case in which respiratory obstruction and fractured ribs, occurring during a sustained epileptic seizure, necessitated tracheotomy. This is possibly one of the earliest cases in which ventilatory distress occasioned by a central nervous system affliction necessitated the operation, an indication later to assume major importance.

McBurney [196] in 1883 described the use of the procedure as a preliminary to certain operations on the head and neck; this use was emphasized by Annandale[3] in 1886, who also reported on tracheotomy as a means of administering anesthesia. In 1904 Larlheujue noted that tracheotomy might be necessitated by accidents occurring during the administration of inhalant anesthesia.[52] In 1906 Cockle[52] advised that the tracheotomy opening be used for the administration of general anesthesia.

Widespread use for diphtheria and croup, and for laryngitis and laryngeal edema, continued to such an extent that by 1887 Lovett and Munro[179] could report 327 cases of diphtheria from the Boston City Hospital treated by tracheotomy. In 1891 Prescott and Goldthwaite[216] reported 392 cases treated by intubation and 139 cases treated by tracheotomy from the same institution. In 1892 Krause[159] also reported on 200 cases of croup and diphtheria in children treated with tracheotomy.

Standardization by Jackson,[152, 153] in the early years after 1900, of both the indications and technic was sorely needed, since errors of judgment and technic were commonplace. With such standardization, however, more and more emphasis was placed on obstruction of the airway at the laryngeal level as the prime need for tracheotomy, which became recognized mainly for its value as a bypass of these physical barriers to ventilation. The indication of mechanical laryngeal obstruction became so well established that Figi,[88] in a review of 200 cases in 1934, and Fitz-Hugh,[89] in a review of 100 cases in 1940, both stated that little new information on this aspect could be added.

With minor exception, many of the writings on this subject in the early twentieth century were concerned, not with the discovery of new indications, but of better recognition of the clinical signs of the established indications, the hazards of improper technic, and the necessity for precise aftercare of the patient.[45, 46, 76, 88, 89, 109, 131, 139, 142, 147] Even today these concepts remain as fundamentals in the proper management of the patient with respiratory obstruction.

As early as 1931 Wilson[291, 292] had mentioned that tracheotomy might be of

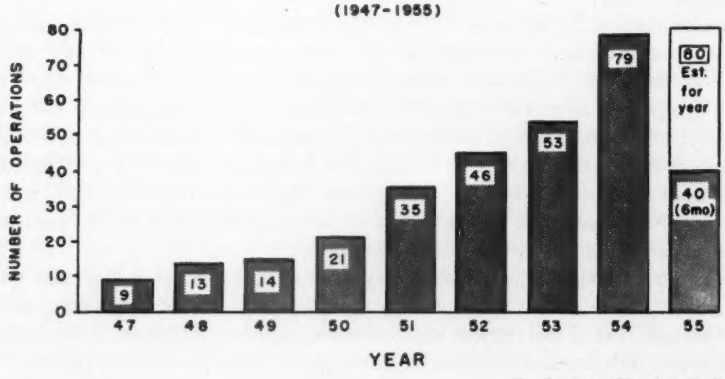
benefit in relieving lower respiratory obstruction, and in 1936 Davidson[70] mentioned tracheotomy in the relief of respiratory obstruction in poliomyelitis. The first actual reporting of results in this use was not done prior to Galloway's work in cases of poliomyelitis in 1943.[99] This study initiated a marked change in medical thought concerning the possible benefits of tracheotomy in cases of secretional ventilatory obstruction from all causes. Since 1943 a succession of reports has appeared describing the use of the procedure for secretional rather than mechanical respiratory obstruction; for patients with poliomyelitis,[2, 34, 67, 99, 103-106, 128, 189, 202, 209, 217, 218, 220] tetanus,[62, 129, 237, 271] barbiturate poisoning,[175] botulism,[26] eclampsia,[57, 58] chest injuries,[40, 41, 42, 13, 289, 290] and head injuries[35, 81, 82] in connection with neurosurgery;[75, 259] and in postoperative pulmonary complications after major surgery.[4, 6, 59, 98, 226] Additional compilations of the general use of tracheotomy in secretional anoxia from all causes have also appeared.[90, 91, 134, 156, 260, 274, 275]

CHAPTER III. CLINICAL MATERIAL

Introduction

Since Galloway's report[99] on the benefits of tracheotomy in bulbar poliomyelitis, there has been a decided change of medical thinking concerning the uses of this operation. Tracheotomy has become well accepted in the management of nearly every type of ventilatory distress. The indications for tracheotomy have increased in scope and number[90] and there are few contraindications to its use.

The present study of the indications, technic, and complications of 310 tracheotomies in 300 patients was undertaken in the hope that such information would be a substantial contribution to the management of these newly recognized respiratory problems and would accurately correlate the various ideas to be found in the current literature. The occurrence of these cases during the period



Brooke Army Hospital, U. S. Army

FIG. 1. The incidence of tracheotomy by year in 310 operations

of time when the newer concepts of the operation were being developed permitted a graphic demonstration of the reasons behind the increased use of tracheotomy.

There has been a steady year-to-year climb in the number of operations performed (figure 1). The chief reason for this has been the recognition of the usefulness of tracheotomy for secretional rather than mechanical ventilatory obstruction. Of the 310 operations, 162 were performed for secretional obstruction and indications based upon a better understanding of the physiopathology of reduced pulmonary ventilation, while 148 were done for mechanical airway obstruction.

In this patient group there was observed nearly every presently known indication for the operation, notable exceptions being for diphtheria and for removal of foreign bodies, two of the earliest reasons for tracheotomy. Certain lesser publicized uses and indications for tracheotomy, particularly in connection with postoperative care following thoracic surgery, severe burns, neck injuries, anesthesia and respiratory resuscitation, were uncovered and will be stressed.

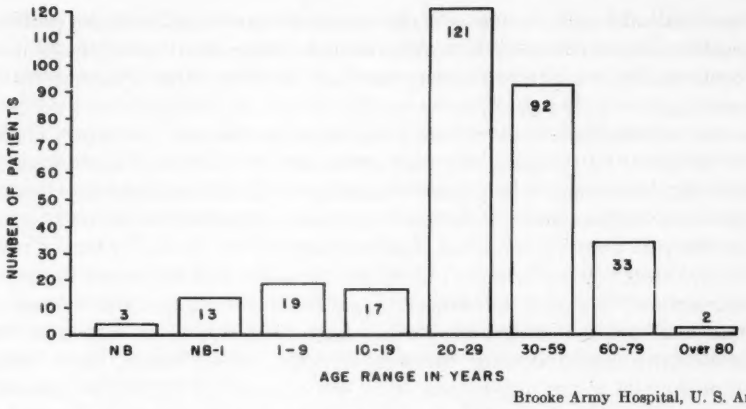
Although recent reports are concerned chiefly with the newer indications and have given little emphasis to the technic of the operation, much can still be said on this aspect of tracheotomy. A wide variety of technics was employed by the various surgeons in these 300 cases, both in emergency and elective operations. In certain instances these technical variations had a direct bearing on the efficient postoperative management of the patient, and on rare occasions even determined whether or not the patient survived. In this respect it was noted again and again that the time spent to excise a portion of the anterior tracheal wall in elective and even urgent tracheotomies was repaid manyfold because of the increased ease of all aspects of aftercare.

Jackson[139, 147, 153] and others[46, 89, 90, 91, 134] have shown that, with reasonable care and a common sense approach to technic and aftercare, the complications and mortality due solely to the operation are negligible. This impression was confirmed in this series despite the application of different technics by many surgeons at various levels of training. The advantages of tracheotomy overwhelmingly outweighed the disadvantages in these patients, and in many instances the timely application of tracheotomy was believed to be the ultimate determinant of survival.

Material

During the period from January 1947 to July 1955 there were 300 patients at Brooke Army Hospital, Fort Sam Houston, Texas, upon whom 310 tracheotomies were performed. These are consecutive, unselected cases and comprise the largest series reported to date which includes mixed indications for the operation.

The range in age of the 300 patients was from the newborn to 82 years of age (figure 2). The three youngest patients were less than 36 hours old at the time of the operation. Over 10 per cent of the patients were under seven years of age. They form an interesting group because of the relatively high incidence of complications and technical difficulties encountered. Forty per cent of the patients were in the age group of 20 to 29 years, not a surprising finding in a study concerned with a large number of armed services personnel. Likewise, the proportion



Brooke Army Hospital, U. S. Army

FIG. 2. The range in age of 300 patients undergoing tracheotomy

of male to female was over five to one. However, the wide range in age and diversity of indications and patient material signifies that this study is quite similar to that which might be encountered at any university medical center.

These operations were performed mainly by staff members and residents in the various surgical specialties, chiefly otolaryngology, general and thoracic surgery, and neurosurgery. An occasional tracheotomy was performed by an intern, internist, or pediatrician. These facts are healthy signs that the indications and technic of the operation are becoming familiar in many fields of medicine and surgery, not just one specialty.

Indications for Tracheotomy

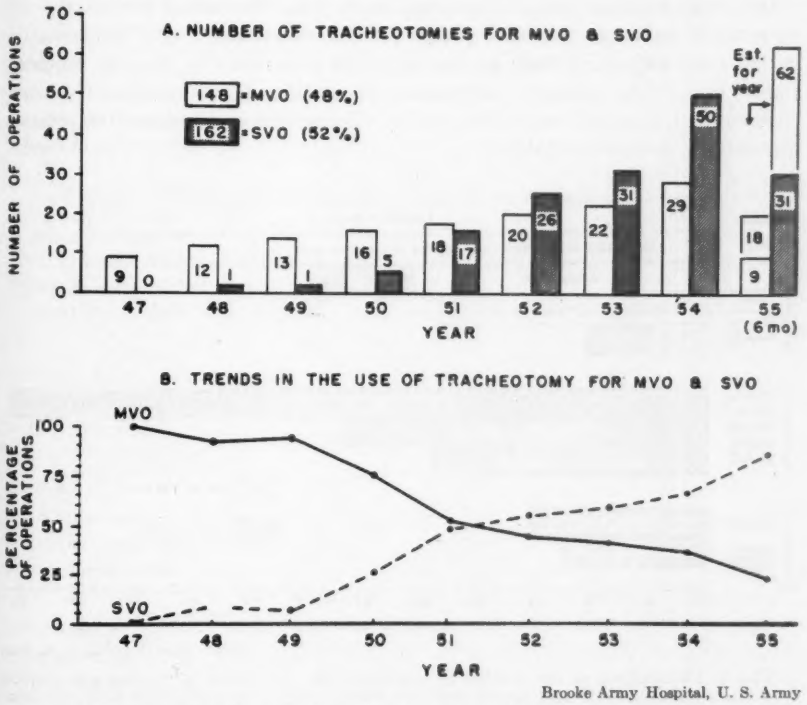
General considerations. Virtually every form of respiratory obstruction, whether due to tumor, trauma, infection, or retained secretions, is mechanical in nature. For the purposes of this discussion the indications for tracheotomy were separated into two broad categories: those due to mechanical and those due to secretional ventilatory obstruction. Such a division is desirable because of certain clear-cut clinical differences in the two types. Mechanical obstruction generally results from narrowing or obturation of the airway. Included in this group are the better known and classical indications for tracheotomy. On the other hand, secretional obstruction usually occurs in association with diseases which may have no direct relationship with the respiratory tract, and the effects of retained secretions may be only one of several manifestations of the primary disease. This group includes the newer, less well known indications for tracheotomy which have become of such great importance lately.

Analysis of the indications for tracheotomy in 300 cases from the combined reports of Figi[88] in 1934 and Fitz-Hugh[89] in 1940 reveals that 296 of the operations were performed for mechanical airway obstruction, chiefly due to diseases of the larynx, trachea, and adjacent tissues.

In a later report of 150 cases, Fitz-Hugh[90] emphasized the increased use and

changing indications of tracheotomy. In these 150 cases 53 per cent had mechanical obstruction, 20 per cent had secretional obstruction, and 27 per cent required tracheotomy for prophylaxis of respiratory obstruction, mostly for mechanical reasons.

In the present study of 310 operations, the increased use of tracheotomy for secretional obstruction is even more decided, with 162 (52 per cent) of the operations being performed for this reason. A comparison of the number of tracheotomies performed each year for mechanical and secretional ventilatory obstruction is shown in figure 3*A*. Although the number of tracheotomies performed each year because of mechanical obstruction has increased steadily in accordance with the over-all increased use of the operation, the increase of operations for secretional obstruction is even greater, thus indicating the recent recognition of the physiological benefits of the operation. This striking change in medical thinking is even more clearly delineated in the trend chart (figure 3*B*), wihch compares the percentage of operations performed each year for each of these two major divisions of indications. Before 1951 there were only scattered instances in which tracheotomy was employed for secretional obstruction, but in



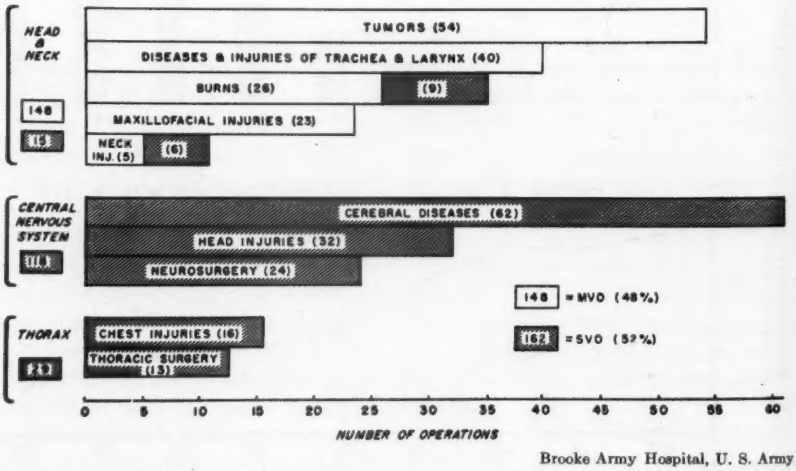
Brooke Army Hospital, U. S. Army

FIG. 3. Comparison of the use of tracheotomy for mechanical ventilatory obstruction (MVO) and secretional ventilatory obstruction (SVO) in 310 operations (1947–1955).

that year and afterward this use became more prevalent, until by 1955 over 77 per cent of these tracheotomies were being done for this reason. The impetus for this change was supplied chiefly by the reports of Galloway[99, 103, 104] and others[8, 34, 111, 207, 217, 218] on the advantages of tracheotomy in poliomyelitis, and by the results at Brooke Army Hospital in 1951 and 1952 in cases of poliomyelitis,[282] head and chest injuries, and in thoracic surgery patients. In 1952 the use of tracheotomy for secretional obstruction surpassed that for mechanical obstruction, and since that time the trend has continued steadily in this direction (figure 3*B*).

In 200 cases reported in 1934 by Figi,[88] over 75 per cent of the tracheotomies were performed for carcinoma or other diseases of the larynx alone. In the present study fewer than one-fourth of the 310 operations were concerned with tumors or other diseases directly involving the larynx. The distribution of the 310 operations according to the primary disease site (figure 4) reveals that 147 of the tracheotomies were concerned with diseases or trauma of the central nervous system or thorax, and 163 tracheotomies were concerned with diseases of the head and neck. The majority of these latter patients had involvement extrinsic to the larynx.

It is then evident from the present study that mechanical ventilatory obstruction is no longer the chief reason for tracheotomy and that the larynx or immediately adjacent tissues are no longer the major sites of diseases requiring tracheotomy. The complete tabulation of specific disease entities requiring tracheotomy, classified as to the primary disease site and cause of respiratory obstruction, is shown in table 1.



Brooke Army Hospital, U. S. Army

FIG. 4. Comparison of the number of tracheotomies performed for mechanical ventilatory obstruction (MVO) and secretional ventilatory obstruction (SVO) in 310 operations: classified as to primary disease site.

TABLE 1

The specific entities causing mechanical and secretional ventilatory obstruction in 310 tracheotomies: classified as to primary disease site

Mechanical Ventilatory Obstruction—148	
Head and neck—148	
Tumors—54	
Presurgery	19
Tumor obstruction	16
Preradiation therapy	8
Radiation therapy obstruction	4
With radical neck dissection	7
Trachea-larynx—40	
Inflammation	15
Following extubation	15
Injury	5
Vocal cord paralysis	4
Congenital obstruction	1
Burns—26	
Head and neck only	10
And other locations	11
And respiratory burn	5
Maxillofacial injuries—23	
With fracture	16
Soft tissue injury alone	7
Neck injuries—5	
Soft tissue injury alone	5
Secretional Ventilatory Obstruction—162	
Head and Neck—15	
Burns—9	
Head and neck and other locations	8
And respiratory burns	1
Neck injuries—6	
Fracture cervical spine	6
Central nervous system—118	
Cerebral disease—62	
Poliomyelitis	35
Encephalitis	7
Cerebrovascular accident	4
Embolism	4
Fat embolism	3
Air embolism	1
Barbiturate poisoning	3
Tetanus	2
Miscellaneous (with associated coma)	7
Uremia (anuria)	3
Septicemia	1
Cirrhosis	1
Diabetes mellitus	1
Porphyria	1
Head injuries—32	
Head injury alone	16
With other major injuries	16

TABLE 1 (Continued)

Neurosurgery—24	
Postoperative coma	9
And respiratory failure	5
Secretional obstruction mainly	10
Thorax—29	
Thoracic surgery—13	
Postoperative secretional obstruction	11
Pulmonary surgery	9
Esophageal resection	2
Relieve closed glottis pressure on bronchial stump	2
Chest injury—16	
"Wet lung"	8
Flail chest	8

Mechanical ventilatory obstruction. Causes for mechanical ventilatory obstruction:—Of the 148 operations for mechanical ventilatory obstruction, 73, or nearly one-half, were performed because of neoplasms, inflammations, and trauma of the larynx and trachea. The balance of the operations in this group resulted from types of obstruction that were not always recognized as requiring tracheotomy until recent years. This latter group includes maxillofacial injuries; burns of the head, neck and respiratory tract; structural alterations from radical surgery of the neck and floor of the mouth, and complications following thyroid surgery.

Effects of mechanical ventilatory obstruction:—The most dramatic respiratory obstruction are those of mechanical origin which develop suddenly and are of profound degree. The medullary centers are strongly stimulated directly from the lowered oxygen tension in the cerebral blood and indirectly through anoxic stimulation of the sinoaortic chemoreceptors.[116] The response is one of marked increase in respiratory movements and frantic efforts to regain ventilation. In a matter of minutes the cerebral and medullary centers become narcotized, and loss of consciousness ensues, followed by cardiorespiratory failure and death. Even if relief is obtained short of death, the higher centers may remain permanently damaged since they are more susceptible to the effects of hypoxia and hypercapnia than the medulla, which is phylogenetically older.[108]

The effects of chronic or partial mechanical obstruction are somewhat different. Insidious hypoxia and hypercapnia develop and alter the chemical respiratory equilibrium so that the medullary centers are affected directly by the altered concentrations of both oxygen and carbon dioxide.[162] The first effort at compensation is an attempt to increase ventilation. In the presence of obstruction this increase in ventilation may not be enough to rid the blood of accumulated carbon dioxide, and other compensatory mechanisms must take place to maintain normal blood pH. The net effect is a compensated respiratory acidosis. The margin of safety under these conditions is quite narrow, and only brief periods of additional hypoxia and hypercapnia are required to upset this delicate balance and produce respiratory failure. These observations were borne out in the group under present consideration, especially in those cases with chronic airway ob-

TABLE 2

The syndrome of mechanical respiratory obstruction

1. Presence of tumor, trauma or disease in direct association with the upper airway
2. Signs of laryngeal dyspnea:
   a. Indrawing at the suprasternal notch, around the clavicles, of the intercostal spaces and the epigastrium
   b. Restlessness, apprehension and disorientation; later coma
   c. Respiratory stridor, rather than wheezing
   d. Choking and waking as the accessory muscles of respiration relax on falling asleep
   e. Pallor; later cyanosis, a dangerously late sign
3. Early—there are obvious signs that the patient is breathing against resistance and not moving enough air
4. Late—fatigue and complete exhaustion

struction due to neoplasms or laryngeal stenosis. If acute obstruction supervened from diagnostic instrumentation, tumor growth or radiation therapy, many of these patients promptly succumbed despite tracheotomy.

Recognition of mechanical ventilatory obstruction:—An unobstructed airway is a clear cut prerequisite to proper ventilation.[23] When this principle is violated by an abrupt mechanical occlusion, the characteristic signs and symptoms demanding relief are usually recognized and energetically treated (table 2). If there is no defect in the respiratory mechanism other than obstruction, it is usually obvious that the patient is working against resistance and not moving enough air. In general, the higher the level of obstruction, *i.e.*, the nearer to the larynx, the more external evidence there is of dyspnea.[110] The classic signs of laryngeal obstruction (table 2) were recognized in 1885 by Montgomery,[191] who made note of the retraction above and below the sternum, above the clavicles, and in the rib interspaces due to the action of the "extraordinary" muscles of respiration, and the marked dyspnea and stridor seen in cases of croup. The clinical importance of these findings has been emphasized by Jackson[147–149, 153] and others.[110, 45, 86, 69, 200] As stressed by Jackson,[147, 105] in practically no other form of dyspnea is such marked retraction noted. The retraction results from efforts by the accessory respiratory muscles to increase the effective negative intrapulmonary pressure and secure more air through the narrowed passage. The presence of stridor or crowing respiration rather than wheezing is stressed by Cawthorne.[45] Cyanosis is a dangerously late and unreliable finding, often masked by pallor, and cannot be relied upon as an early or, in some instances, even late sign of respiratory obstruction. Excitement, restlessness, agitation, and extreme apprehension are more constant findings. Kully[163] states that the patient's feeling of impending doom is reflected even in the facies of infants. The failure to recognize the cause of the restlessness and the administration of sedatives empirically is criminal.[142, 147] Unrelieved obstruction eventually leads to fatigue and complete exhaustion. The subdued behavior of the patient from weariness must not be interpreted as a sign of improvement. This is a danger signal indicating that unless obstruction is relieved soon, the patient will be lost.

Treatment of mechanical ventilatory obstruction:—The only two satisfactory methods for treatment of mechanical airway obstruction are removal of the

obstruction or bypassing it. On occasion, a combination of these two methods is indicated. With most mechanical obstructions, especially those of sudden or profound nature or those of chronic fixed type, it is best to assure the airway by tracheotomy, initially. More leisurely steps can then be directed at the obstructive lesion. In a few cases initial endoscopic treatment might be justified. If unsuccessful, however, such methods may intensify the obstruction and increase the danger of asphyxia. Several urgent tracheotomies in this series of patients were occasioned following diagnostic or therapeutic instrumentation of this sort. In many cases it is desirable to insert a bronchoscope beyond the obstruction, so that tracheotomy becomes less of an emergency.[109, 173, 270] In the group of patients under present consideration, this method of assuring the airway so that deliberate tracheotomy could be done was used frequently. Again, it should be pointed out, on occasion the patient was made worse by unsuccessful attempts at bronchoscopy. The degree of obstruction, availability of equipment, and skill of the endoscopist are factors that must be considered carefully in decisions of this sort.

Permanent obstructions below the usual tracheotomy site are difficult to treat by any means. Tracheotomy will be of little value unless a long cannula can be passed beyond the obstruction, as described by Jackson[139] and Rethi.[225] Temporary relief can be obtained by passing a bronchoscope or endotracheal tube beyond the obstruction, but such measures are poorly tolerated by the patient for more than short periods. Temporary obstructions below the usual tracheotomy level, such as foreign bodies, are best approached by bronchoscopy or, if necessary, bronchoscopy through the tracheotomy opening. The use of a tracheotomy opening alone for access to foreign bodies has been completely discarded as a satisfactory means for the removal of foreign bodies.

Secretional ventilatory obstruction. Normal control of secretions:—Secretions in the respiratory tract are formed in a methodical manner and removed unobtrusively by several intrinsic respiratory mechanisms, the most important of which are ciliary activity and a proficient cough reflex.[23, 153] Positional change and postural drainage are important adjuvants in removing accumulated secretions.[176] Bronchial peristalsis, which is a slow, rhythmic movement of the bronchial musculature from the periphery toward the hilum, undoubtedly assists in maintaining a clear airway.[9, 23, 176] Ciliary action is an important means of propelling secretions along the respiratory tree, but its effectiveness is quickly reduced or eliminated by drying or by changes in the viscosity of the bronchial mucus from atropine, potassium iodide, and anesthetic agents.[205]

The importance of the cough reflex was recognized years ago by Jackson,[140, 145] who considered the "tussive squeeze" to be the most important means by which distal secretions are milked into the larger bronchi within range of the coughing blast or bronchoscope. The normal cough reflex consists of inspiration, closure of the glottis, an increase in intrathoracic pressure, opening of the glottis, and a sudden expiration, with release of the pressure.[190, 255] Weakening or abolishment of the cough reflex is the single most important factor in respiratory obstruction from retained secretions.[141, 275]

Causes for secretional ventilatory obstruction:—Although there are many

TABLE 3

Causes for loss of the cough reflex

I. Patient *unwilling* to cough
  A. Pain
    1. Incisional
    2. Chest injury
II. Patient *unable* to cough
  A. Loss of conscious control
    1. Anesthesia
    2. Coma from any cause
    3. Debilitating diseases
  B. Weakness or paralysis of the intrinsic mechanism
    1. Poliomyelitis or other cerebral diseases
    2. Trauma or operations on the brain and spinal cord
    3. Impairment of the thoracic bellows system

different disease states that may disrupt the normal control of secretions (table 1), the basic deficit in all these lesions is dysfunction of the cough reflex. The mechanisms by which coughing is blocked (table 3) may be conveniently separated into two groups of patients—those who are unwilling to cough and those who are unable to cough.[289] Pain on movement of the chest wall is the chief reason for unwillingness to cough. This occurs most frequently from thoracic or abdominal incisional pain[23] and in chest injuries, especially those with rib fractures.[289] The injudicious use of narcotics does little to promote spontaneous coughing.[23, 141] Strong encouragement and assistance are necessary if this type of patient is to clear his airway voluntarily. About 15 per cent of all secretional obstructions in the present series was due to unwillingness to cough because of pain. Inability to cough is due most frequently to anesthesia, coma from any cause, paralyses, and debilitating diseases. In these instances the anatomic mechanism for coughing may be intact, but the central response to stimulus depressed. Accumulated secretions must be removed for the patient if a clear airway is to be gained. Almost 55 per cent of patients with secretional obstruction were unable to cough for this reason. In other cases of inability to cough, the fundamental deficit is a disruption of the neuromuscular pathways for coordinated movements of the glottis and pharynx, or weakness of the muscles necessary to increase intrathoracic pressure, or both. The best known example is poliomyelitis, which may interrupt either or both of these mechanisms. Again, secretions must be removed for the patient. About 30 per cent of the cases had secretional obstruction due to this cause.

Additional factors in the development of secretional obstruction are overproduction of secretions, thickening of the secretions, and ingress of fluid not normally present in the respiratory tract. An increase of bronchial secretions and thickening of bronchial mucus are induced following blunt trauma to the chest wall.[74] In addition, bronchospasm is produced, and the stage is set for secretional obstruction and atelectasis. Bronchorrhea is seen following respiratory burns, and after inhalation of chlorine or phosgene gas. In tracheobronchitis, or bronchiectasis, an excess of thick secretions is already present. If the cough reflex is

then weakened or abolished, secretional retention and obstruction may ensue rapidly. Fluids not normally present in the tracheobronchial tree, such as vomitus or saliva, may be aspirated during or after anesthesia,[4] or because of a poorly coordinated swallowing mechanism, such as in poliomyelitis.[104, 105]

Effects of secretional ventilatory obstruction:—The deadliness of accumulated secretions in the respiratory tract has long been recognized. It is clear that Montgomery,[191] who in 1885 advised aspiration of secretions in the trachea before inserting the tracheotomy cannula, also recognized certain of the dangerous chemical changes that occur in the blood stream during airway obstruction when he stated: "The want of oxygen, and increase of carbonic acid, engenders blood decomposition, so grave in character, that even, although air is again permitted to enter the lungs freely . . . the relief is but temporary, and the rekindled flame is . . . finally extinguished."

In recent years the physiological abnormalities that result from secretional obstruction have been carefully studied.[101, 102, 104, 116, 212, 264, 275] These abnormalities, which consist of hypoxia, hypercapnia, and acidosis, are collectively referred to as asphyxia.[275]

Kubicek and his collaborators[161, 162] have pointed out that only a small decrease in oxygen saturation of the arterial blood may be reflected as a marked diminution in the gaseous tension of oxygen in the tissues. This tissue hypoxia causes increased capillary permeability, pulmonary edema, and damage to nerve and cardiac tissues. The narrow margin of safety during periods of oxygen want has been stressed by Galloway[101] and others.[25, 264] Thorner and Lewy[264] believe that even one minute of oxygen deprivation may be followed by morphologic changes in the brain. Others[61, 175, 293] have shown that three to eight minutes of oxygen lack is enough to produce widespread damage, irreverisble changes, and death. Even brief periods of hypoxia, especially if repeated, may cause lasting mental changes, even though the patient survives.[101, 275]

Of equal importance is the retention of carbon dioxide in the blood as free carbonic acid. Because of its greater molecular weight, carbon dioxide diffuses less readily than oxygen and although because of solubility factors it crosses the alveolar membranes more rapidly than oxygen, under conditions of reduced alveolar gaseous exchange it is retained in the blood.[275] Hypercapnia produces headache, restlessness, uncooperativeness, and disorientation in its early stages, while high concentrations induce narcosis, respiratory depression, and circulatory collapse. The administration of oxygen, while of the greatest importance, will not, by itself, substantially affect the carbonic acid blood level if the mechanism for respiratory obstruction remains uncorrected.[31, 212] Blalock and his associates[25] have shown experimentally that hypercapnia may reach dangerous levels in the presence of respiratory obstruction, while oxygen saturation is still satisfactory. This impression has been confirmed clinically by several observers.[31, 212, 275] Bower and his associates[31] found that many poliomyelitis patients were in profound states of respiratory acidosis due to insufficient eradication of carbon dioxide, which occurred despite the offering of additional oxygen. Systemic attempts at correction were found to be valueless unless secretional obstruction

was alleviated. Acidosis develops not only from the hypercapnia but is accentuated by the production of excess lactic acid in the tissues as a result of oxygen lack. The severe acidosis that results is of combined respiratory and metabolic origins,[275] and if the respiratory obstruction is not corrected by mechanical means, other therapeutic efforts will be largely valueless.

Many vicious cycles that intensify and promulgate the alterations of respiratory physiology may occur in the presence of secretional obstruction.[275] In the lungs alone, hypoxia may cause increased capillary permeability, resulting in pulmonary edema and further hypoxia. Increased respiratory efforts may transmit venous pressure elevations to the cranial cavity and depress the central nervous system. With prolonged or severe hypoxia and hypercapnia, the respiratory center may become depressed, reduce the respiratory efforts, and lead to further asphyxia. In the presence of a damaged central nervous system either from trauma or disease, such as poliomyelitis, encephalitis, or barbiturate poisoning, these effects are intensified to the maximum. Increased carbon dioxide content of the blood is a potent vasodilator of the cerebral vessels which may increase central nervous system depression by the resulting increased pressure and edema. The increased capillary permeability from hypoxia may increase cerebral edema, and even induce renewed bleeding from traumatized vessels. It is also believed that increased intracranial pressure, because of increased right atrial and pulmonary pressures, may cause an outpouring of pulmonary secretions, leading to pulmonary edema.[78]

Recognition of secretional ventilatory obstruction:—Respiratory obstruction may develop suddenly due to an abrupt influx or accumulation of secretions, but these instances are uncommon. In such cases, the volume of secretions necessary to produce rapid obstruction usually produces obvious signs that such an excess is present. More often secretional obstruction is a devious and insidious intruder which is easily overlooked in the press of matters concerning the primary disease. It is now well known that a wide variety of pathologic conditions may have associated secretional obstruction which can be just as deadly as the primary lesion (table 1).

The first step in the diagnosis of secretional obstruction is to recognize that disease states or trauma are present that may interrupt normal coughing or the coordinated functions of respiration. The next step is the recognition that certain systemic or local manifestations might be the result of blockage in the lower respiratory passages (table 4). The most reliable findings are those related to the lungs, such as rhonchi, a wet cough, and physical or roentgenographic evidence of atelectasis. Frequently these findings may be minimal or absent, with the only changes being alteration of the vital signs plus apprehension, restlessness, and irritability. All present-day observers[162, 202, 212] emphasize that cyanosis is an unreliable indicator as to the presence and degree of cellular oxygen want, and that if cyanosis is used as a criterion for initiating treatment for hypoxia, extensive nervous system damage may have already occurred. Elevations of pulse and temperature and irritational states are much more sensitive indicators of reduced oxygen tension in the tissues.[162]

TABLE 4

The syndrome of secretional ventilatory obstruction

1. Presence of disease, trauma or surgery that obtunds the natural cough reflex:
   a. Diseases producing coma or respiratory weakness of either central or peripheral origin
   b. Neurosurgical operations or head injury
   c. Thoracic surgery or chest injury
   d. Any major operation
2. The patient is unwilling or unable to cough effectively from any of the above causes
3. Cough, if present, is rattling, "wet," and does not completely clear the respiratory tree
4. Increased respiratory efforts early—decreased respiratory efforts later or if primary disease depresses or weakens the normal respiratory mechanism
5. Increased pulse, blood pressure and temperature—later circulatory failure
6. Mental symptoms from hypoxia and hypercapnia
7. Clammy, perspiring facies, occasionally cyanosis
8. Exhaustion
9. Physical and roentgenographic findings of "wet" or congested lung, atelectasis, or pneumonitis

There are two chief reasons for failure to recognize secretional obstruction of the lower airway. The first is that the possibility of secretional obstruction is disregarded just because obvious signs of respiratory obstruction are not present. In many of these disease states, the patient's ability to manifest obvious external signs of respiratory obstruction is nullified by central respiratory depression or weakness of the respiratory muscles. Secretional obstruction may have to be diagnosed on suspicion alone.[244] Frequently such obstruction may be suspected when normal therapeutic efforts fail to produce the expected response of the patient. In the present series of patients with head injury and severe central nervous system disease, unrecognized secretional blockage was often the cause for continued symptoms. In certain of these cases, even though there were no demonstrable signs of accumulated secretions, copious quantities of thick mucus could be obtained after tracheotomy, and the patient often improved.

The second reason for failure to recognize secretional obstruction is the tendency to blame the signs and symptoms on the primary lesion. Since the findings from secretional obstruction are frequently identical with those of the primary lesion, it may be difficult to be certain that any untoward reaction of the patient is due solely to his disease process. Only when the airway can be guaranteed to be clear, by tracheotomy if necessary, can this question be settled.

Treatment of secretional ventilatory obstruction:—Jackson,[141, 143, 146, 148] Leopold[171] and Lee[166-169] were the first to show that "pneumonia" developing after operations or in association with a number of medical diseases was, in reality, commonly atelectasis. Lee[167, 168] clearly demonstrated that atelectasis was not related to antecedent bacterial invasion but could be explained on a purely physical basis from two associated causes: (1) loss of the cough reflex and, (2) retention of secretions.

Once the mechanism for secretional obstruction was understood, rational methods of treatment evolved. The first of these was the "stir-up" regime,

which consisted of frequent changes of body positions, the use of postural drainage, and encouragement of coughing and deep breathing by the patient.[77] The value of preservation of the cough reflex by limiting suppressives and of catheter suctioning of the mouth and hypopharynx also became recognized. In certain cases, however, these methods were found to be inadequate because of limited cooperation on the part of the patient. For this reason more active measures for aspiration of retained secretions became advocated. These included intermittent intratracheal catheterization, an indwelling intratracheal catheter, bronchoscopy, and tracheotomy.

The advantages of intermittent intratracheal aspiration with a catheter are that it is simple, it can be repeated as necessary, and it can be employed by nursing personnel.[39, 123, 124, 239] Because of these advantages intratracheal aspiration has become a popular substitute for bronchoscopy in the management of secretional obstruction. In this group of patients, however, certain shortcomings were noted. Contrary to popular belief, the passage of the catheter does require a degree of skill, and it requires cooperation of the patient. In addition, it is difficult to obtain thorough cleansing of the respiratory passages, and the frequent repetitions of the process may cause trauma to the nasal cavity and larynx, exhaustion, and even death. The experiences in this group of patients revealed that it was not always possible to catheterize the trachea repeatedly with unfailing success, nor was it possible, in certain instances, to accomplish catheterization immediately if sudden or intense obstruction occurred. Intratracheal aspiration was found to be best suited to those patients in whom a limited number of aspirations might relieve the condition permanently.

To avoid certain disadvantages of intermittent tracheal aspiration Buyers[37] has advocated an indwelling double-lumen catheter which can be employed for simultaneous suction and administration of oxygen. The inherent disadvantages of this method are the same as when using an intubation or endotracheal tube.[80] The incidence of spasm and edema of the larynx, glottis, and vocal chords is increased and the danger that laryngeal obstruction might follow removal of these types of tubes must be recognized. In addition, all types of indwelling tubes are subject to being coughed out by the patient, possibly at an unfortunate time. Finally, aspiration of vomitus and fluids administered by mouth is made easier because normal closure of the glottis is not possible with the tube in place.

Bronchoscopy has long been advocated by Jackson and others as the most efficient means of tracheobronchial toilet. In fact, Jackson in 1939[150] stated that bronchoscopy would probably obviate the need for tracheotomy for the removal of secretions. However, the added risk to the patient, the skill required in performance of the procedure, and the harm from repeated instrumentation, all preclude widespread use of bronchoscopy except in well-trained hands.

Tracheotomy should be employed for the relief of secretional obstruction in those patients in whom it is anticipated that short-interval aspiration over a prolonged period will be necessary, and in whom other methods have been tried and found to be inadequate. The advantages of tracheotomy are that it permits easy aspiration of secretions and also permits the administration of high concen-

trations of oxygen directly into the respiratory passages. In addition, tracheotomy bypasses a possible source of obstruction at the larynx, and by diminishing the amount of dead air space decreases the respiratory effort.[40] Contrary to previous belief,[45, 109, 139] it has been found that tracheotomy does not increase the amount of nursing care required for the patient. Indeed, tracheotomy care can be more easily learned and carried out by semiskilled help than any other method of treating secretional obstruction. The disadvantages of tracheotomy are the added risk of any operation plus the complications peculiar to tracheotomy alone. Virtually all these dangers can be avoided if the tracheotomy is performed as an elective procedure and diligent aftercare is employed.

Clinical Cases with Mechanical Ventilatory Obstruction

Tumors of the head and neck (54 cases). The types of tumors and their site of involvement in these cases were: (1) Larynx: malignant, 30; benign, 3. (2) Neck: metastatic, 15; lymphoma, 2. (3) Miscellaneous: pharynx, 2; tongue, 1; tonsil, 1.

The exact reason for the performance of tracheotomy was of interest. These causes were: (1) with laryngeal surgery, 19; (2) with radical neck dissection, 7; (3) obstruction from tumor, 16; (4) prior to radiation therapy, 8; and (5) obstruction during radiation therapy, 4.

Nearly every patient with carcinoma of the larynx may need a tracheotomy whether treatment consists of surgery or radiation. In the 30 cases of carcinoma, 23 patients were treated by surgery and 7 patients were treated by radiation therapy.

In the 15 cases of metastatic carcinoma, the primary sites of tumor involvement were the lip, tongue, larynx, pharynx, and the thyroid gland. Eight patients received radiation therapy, while seven were treated by surgery. Tracheotomy seldom will be required after the usual radical neck dissection unless complications arise. In these seven cases, however, the neck dissection involved more than the usual operation and consisted of a bilateral procedure or partial resection of the mandible, tongue, or floor of the mouth. In these instances the structural alterations are such that collapse of the soft parts about the airway may be anticipated. The necessity for reliable airway following these composite operations has been stressed by Hayes Martin[183, 184] and others.[95, 170]

There are distinct advantages in performing tracheotomy prior to surgery of the larynx or radical surgery of the neck.[88, 89, 95] In this group of patients the advantages noted were: (1) intubation is easier and there is no need for additional manipulation of the anesthesia connections during surgery; (2) operating time during the major procedure is reduced; and (3) the patient becomes accustomed to the cannula. Figure 5 shows a patient with recurrent carcinoma of the lip involving the right mandible. Radicle neck dissection has already been done. The patient is shown at the time of tracheotomy, three days before hemimandibulectomy was performed. This is the type of patient in whom tracheotomy is of the greatest value in protecting the airway following radical surgery on the head and neck.

In 16 patients tumor obstruction necessitated tracheotomy. Most of these

FIG. 5. Patient with recurrence of lip carcinoma in right mandible. Tracheotomy is to be performed before right hemimandibulectomy. Radical neck dissection has already been done. Prophylactic tracheotomy is mandatory in this type of case because of the structural alterations about the airway that result from radical surgery.

patients had chronic airway obstruction and increasing difficulty with ventilation. In three patients apnea occurred after tracheotomy due to a sudden "washing out" of carbon dioxide accumulated during the prolonged period of restricted ventilation. In two patients the apnea responded to rebreathing and artificial respiration, but in one patient the apnea persisted, and despite vigorous attempts at resuscitation death occurred. In six other patients with chronic airway obstruction, acute obstruction supervened because of tumor growth, instrumentation, or radiation therapy. Tracheotomy was urgently required in each of these cases. There were three deaths in these six cases. This emphasizes the fact that patients with chronic airway blockage do not tolerate even brief periods of additional obstruction.

There were nine hospital deaths in this group of 54 patients, a case fatality rate of 15 per cent. Three of these deaths could be attributed partially to errors of judgment in the use of tracheotomy and one death resulted from apnea following the operation. In two cases there was a rather prolonged operation due to the presence of a tumor mass.

The findings in this group of patients confirms the beliefs of others regarding the use of tracheotomy in the treatment of head and neck tumors.[88, 95, 170, 183, 185, 204] First, tracheotomy has decided advantages if performed before extensive head and neck surgery. Second, patients with chronic airway obstruction should have tracheotomy performed before acute obstruction supervenes. Such acute obstructions may be expected at any time from the growth of the tumor, but

may be especially anticipated where instrumentation or radiation therapy is to be employed. Third, apnea due to washing out of carbon dioxide accumulated during chronic respiratory obstruction should be expected following tracheotomy in a substantial number of cases and should be met by artificial pulmonary ventilation.

Diseases and injuries of the trachea and larynx (40 cases). The causes for mechanical ventilatory obstruction in this group of 40 cases were: (1) inflammation, 15; (2) following instrumentation, 15; (3) trauma to larynx or trachea, 5; (4) vocal cord paralysis, 5; and (5) congenital obstruction, 1.

Inflamation (15 cases):—The diseases responsible for tracheotomy were laryngotracheobronchitis in nine cases and angioneurotic edema, acute laryngitis, and tuberculous laryngitis, in two cases each.

Laryngotracheobronchitis is one of the earliest diseases in which there was recognized the need for removal of secretions as well as the need for bypassing an obstruction.[110] However, this important concept of relieving secretional obstruction was not recognized in other diseases until lately. The incidence and severity of this disease have lessened in recent years. This is due primarily to earlier, more adequate treatment of all respiratory infections. The mortality reported in these cases up to 1940 was 50 to 60 per cent.[89, 163, 227, 228] Recent reports have shown fatality rates ranging from 2 to 9 per cent.[86, 200]

Better methods of treatment of acute laryngotracheobronchitis with cool vapor and oxygen[71] and antibiotics have reduced the number of tracheotomies necessary in this disease. All nine tracheotomies were performed before 1950, and since that time tracheotomy has not been required once in this disease. In those patients needing an artificial airway, the treatment of choice is tracheotomy. Intubation tubes are now recognized as being unreliable airways with inherent dangers and complications. All nine of the patients were under six years of age and most were less than two years of age. There were no deaths in the group. In two patients, both dyspneic infants with marked inspiratory retraction, emergency tracheotomy was required. Each of these patients developed mediastinal emphysema and pneumothorax, but the complication was recognized, treated, and did not hinder recovery. In four other patients bronchoscopy was used to maintain an airway while tracheotomy was instituted, thus converting the operation to a deliberate rather than an emergency procedure. Bronchoscopy was not required once following tracheotomy. This is in marked contrast to earlier reports of treatment in laryngotracheobronchitis in which bronchoscopy for removal of mucus and crusts was required frequently.[50, 148] Crusting of tracheal secretions was a problem in two cases, but responded satisfactorily to gentle irrigation and aspiration through the cannula.

Following instrumation (15 cases):—Laryngeal edema and spasm necessitated tracheotomy following diagnostic and therapeutic instrumentation of the following types: (1) bronchoscopy, 8; (2) endotracheal intubation, 4; (3) laryngoscopy, 2; and (4) esophagoscopy, 1.

Many of these patients were children, or adults with preexisting disease of the larynx or esophagus. In some cases the damage to the larynx could be attributed

to prolonged or traumatic instrumentation; in others, laryngeal obstruction followed a short, gentle examination. There were two deaths in this patient group. Both followed bronchoscopy. In one of these cases delay in instituting tracheotomy was partially responsible for death. The other patient died as a result of an unrecognized pneumothorax following tracheotomy. In all other cases tracheotomy proved to be a reliable and prompt means of securing relief from airway obstruction following instrumentation.

Injury to the trachea or larynx (five cases):—Three of these injuries were due to knife wounds while two were caused by wounds from small caliber missiles. In all these cases the damage was limited chiefly to the respiratory passage, although one gunshot wound also caused perforation of the esophagus. In each case the injury to the airway was obvious because of leakage of air into the wound. In none of these patients was respiratory distress of a severe nature. The accepted principles in treatment of trauma to the airway were followed in most of these cases. The wounds of entrance and exit were carefully debrided and closed in layers. Elective tracheotomy was then performed below the site of injury. In one patient closure was performed without tracheotomy. Within a week the patient developed a wound infection which produced respiratory embarrassment and required tracheotomy for relief. In none of the patients was there an attempt to use the wound as a site of tracheotomy, although in one patient the location was suitable. The use of the wound site for tracheotomy usually predisposes to infection, difficulty from an ill-fitting cannula, or damage to the cricoid cartilage if the wound location is adjacent to this structure.

Vocal cord paralysis (four cases):—In these four cases disease or injury to the to the recurrent laryngeal nerves was the precipitating factor of respiratory distress. In two patients paralysis occurred following thyroidectomy and in one patient following radical neck dissection in association with total thyroidectomy. The fourth patient developed bilateral vocal cord paralysis as a result of an ascending paralysis of the Guillain-Barre type.

Burns (35 cases). In the analysis of the broader indications for tracheotomy two distinct types of burn cases were recognized. In 26 patients the burn was of the head, face, neck or respiratory passages, as well as other parts of the body, and produced mechanical ventilatory obstruction (figure 6). In nine patients, extensive burns, mainly of the trunk and extremities, were responsible for inability or unwillingness to cough and resulted in secretional ventilatory obstruction. For clarity in discussion, the entire 35 cases will be considered together.

According to Artz,[5] there are mixed feeling regarding the necessity for tracheotomy in burned patients; in particular, burn treatment centers in Europe rarely use the operation. Even in this country there are relatively few reports on burns which mention tracheotomy.[14, 134, 172] In this group of patients, however, the operation has been considered invaluable to bypass edema and swelling of the glottis or soft tissues about the airway, to remove secretions resulting from the marked bronchorrhea that follows thermal injury to the respiratory mucosa, and to relieve the patient of retained secretions when there is dysfunction of the

FIG. 6. Patient with severe burns of head and neck. Tracheotomy is of great benefit in burn cases, not only to relieve immediate obstruction of the upper airway, but also for the administration of anesthesia during surgical reconstruction later. The use of a face mask for anesthesia in this type of case is dangerous and may convert partial burns into full-thickness burns from pressure of the mask or the irritant effect of the anesthetic agent.

cough reflex. In addition, the tracheotomy may on occasion serve as a valuable means of giving general anesthesia for the operative treatment of these cases.[1, 6]

The average amount of body surface burned in these 35 patients was 40 per cent. There were 16 deaths in this group, and of these, 12 patients had second- and third-degree burns of from 60 to 90 per cent of the body surface. Tracheotomy, used in conjunction with the other energetic measures for treatment of severe burns, was often a vital factor in the initial survival of these patients.

There were two rather tragic cases in which errors in the management of tracheotomy contributed materially to the death of the patient. In each case an ill-fitting cannula was responsible. In the first patient, a 2-year-old child, the tracheotomy tube was too long and eroded through the trachea at the carina (figure 7). The resulting mediastinitis was a contributing factor in the death of this severely burned child. In the other patient, a young man with burns of the face, neck, and respiratory passage, tracheotomy was required because of edema of the glottis. Unfortunately, the cannula used was too short to allow for the swelling of the neck. During the act of swallowing the tube became dislodged into the soft parts of the neck and the patient asphyxiated before the airway could be restored. At autopsy it was determined that only the tip of the cannula had been within the tracheal lumen. This case points out two glaring errors in management. First, where swelling of the neck is present or anticipated a tracheotomy cannula of sufficient length must be employed to allow for the increased

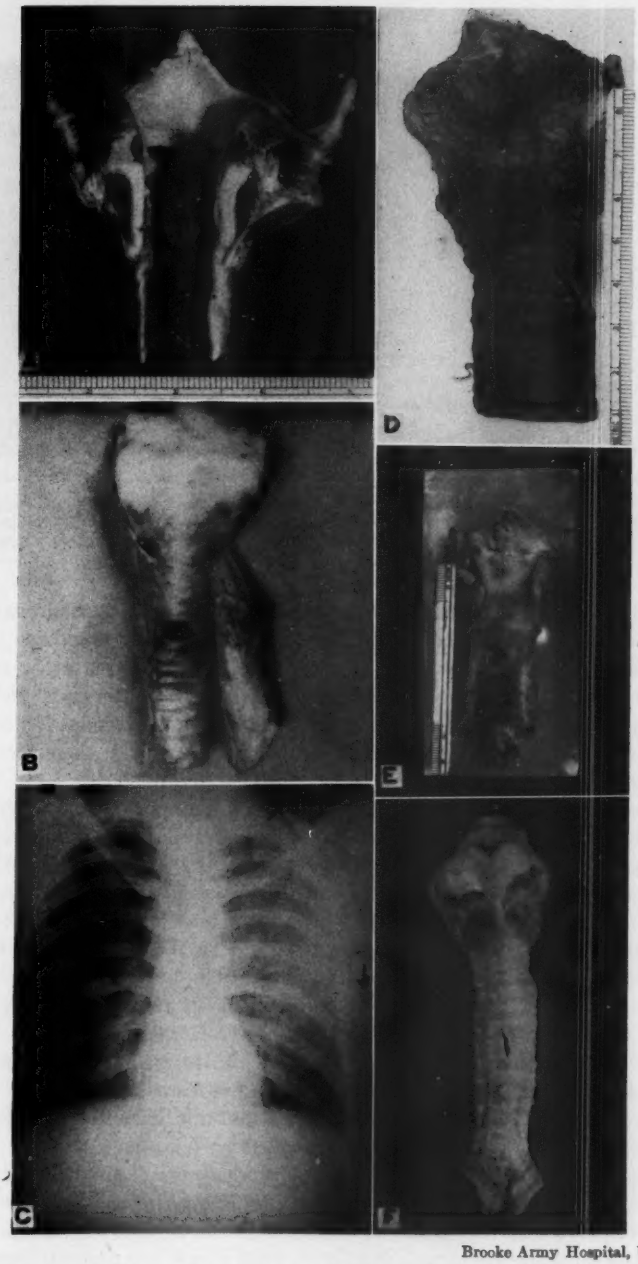


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FIG. 7. The complications of tracheotomy: (A) Mucus plug which obstructed the airway following removal of cannula resulting in death of the patient. (B) Laceration of the esophagus. (C) Pneumothorax, right. (D) Poorly placed tracheotomy opening which made postoperative management difficult. (E) Erosion of trachea and carina from the use of a cannula that was too long. (F) Tracheotomy opening too low for use with a respirator.

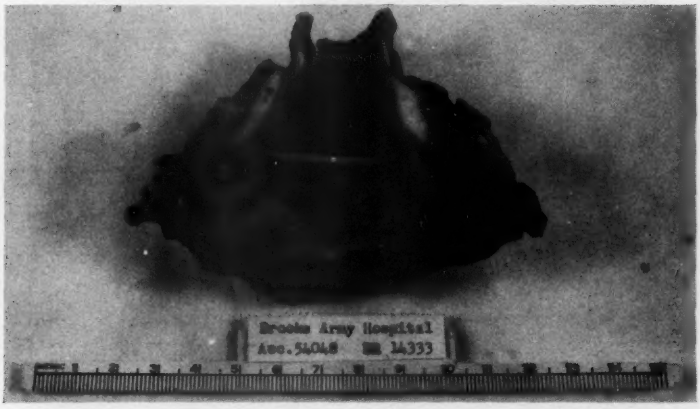

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FIG. 8. Larynx of patient with burns of face, neck, and respiratory passages. Note marked edema of glottis and hemorrhagic inflammatory process of inner larynx and trachea from thermal injury. The patient asphyxiated after his tracheotomy cannula accidentally became dislodged. At autopsy the glottis was completely shut.

depth of the trachea. Second, if at the time of tracheotomy, which was not an emergency in this instance, a piece of the anterior tracheal wall had been excised, retraction of the soft parts might have allowed sufficient respiratory exchange until the cannula could have been replaced. The vertical slit employed in this case closed promptly once the tracheotomy tube became dislodged. Ventilation through normal channels was impossible because of marked edema around the glottis, which was found tightly closed at autopsy (figure 8).

In one patient mediastinal emphysema and minimal pneumothorax developed after tracheotomy. No treatment was necessary, and recovery was not impaired.

Maxillofacial injury (23 cases). Despite over 10,000 recorded instances of maxillofacial injury during the Civil War, tracheotomy was not used once in the surgical management of these cases.[187] The benefits of tracheotomy in maxillofacial injuries were only partially recognized during World War I. As noted by McGee[199] the operation was avoided if at all possible; but once tracheotomy was done, surgical management and the administration of anesthesia were simplified greatly. In World War II casualties the value of tracheotomy, not only for averting asphyxia and giving anesthesia, but also for protection of the airway postoperatively, was clearly recognized for the first time.[94, 210]

The maxillofacial injury was the only significant disability in these 23 patients. There were facial bone fractures in 17 patients, while seven patients had soft tissue injury alone. The majority of these injuries were of the "stoved-in face" type,[210] the result of automobile accidents. This type injury usually does not present the severe threat to the airway as does the penetrating missile wound, seen mainly in battle casualties. This latter type injury was present in only three cases in this group. There were no deaths in these 23 patients, although four patients, when first seen, had extreme respiratory distress and required emer-

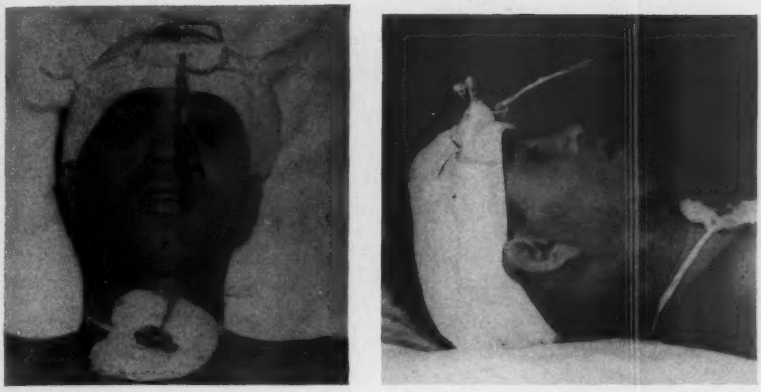

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FIG. 9. Patient with severe maxillofacial injury with multiple fractures of maxilla and mandible. Note the "stoved-in" appearance of face on lateral view. This patient subsequently underwent five major reconstructive operations using the tracheotomy opening for administration of anesthesia.

gency tracheotomy. One of these patients developed pneumothorax following tracheotomy, which was treated successfully by thoracotomy tube and underwater seal drainage (figure 7).

The advantages of using the tracheotomy opening for general anesthesia is clearly shown in this group of patients. There was a total of 43 major operations in these 23 patients, exclusive of tracheotomy. Endotracheal anesthesia was given 36 times through the tracheotomy opening. Some patients underwent as many as five separate corrective procedures (figure 9). The usual method employed for general anesthesia was to spray the trachea with a topical anesthestic, then replace the cannula with a short endotracheal tube. Again the advantages of excising a portion of the tracheal wall were demonstrated. In those patients in whom excision had been done it was no problem to replace the cannula with an endotracheal tube. In a few patients in whom a vertical slit was made in the trachea extreme care had to be taken not to lose the airway during transfer of the tubes.

In the immediate postoperative period following treatment of maxillofacial injuries, tracheotomy is essential to protect the airway in the presence of soft tissue swelling, bulky dressings, and fixation of the mandible and maxilla.

Neck injury (11 cases). Neck injury may result in mechanical airway obstruction from swelling of the soft parts about the airway, or it may cause secretional ventilatory obstruction due to respiratory weakness and inability to cough if the spinal cord is damaged. In six of these patients fracture of the cervical spine caused spinal cord injury and paraplegia. This injury results in a decreased respiratory exchange and inability to develop sufficient intrathoracic pressure to produce a satisfactory cough. The patient not only cannot clear his pulmonary secretions properly, but also is subject to aspiration of food and vomitus. One

patient required emergency tracheotomy because of blockage resulting from aspirated vomitus. Tracheotomy kept the airway clear and improved the efficiency of tidal air exchange. This particular indication for tracheotomy has not been previously described except in one report.[290]

In the other five patients mechanical airway obstruction was the result of hemorrhage and hematoma formation in the soft tissues of the neck following trauma. In one case a persistent tracheotomy fistula required operative closure four weeks following decannulation. This was the only complication of this nature in the entire 310 operations. There were three deaths in this patient group, two the result of spinal cord injury and one from uncontrollable hemorrhage after a gunshot wound involving the carotid vessels.

(*To be continued*)